Murray E. Fowler and Susan K. Mikota

# Biology, Medicine, and Surgery of Elephants

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Murray E. Fowler
Susan K. Mikota

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Murray E. Fowler
Susan K. Mikota

# Introduction

Murray E. Fowler and Susan K. Mikota

Elephants are possibly the most well-known animal in the animal kingdom. The enormous size, unusual anatomy, and longevity of elephants have fascinated humans for millennia. Today, their intelligence, strong family bonds, and the irresistible appeal of their young continue to endear them to many.

Elephants have served man as a means of transport, a vehicle for carrying soldiers into war, and laborers in the timber industry. Despite the long association of elephants with man they have never been truly domesticated.

Elephants evoke strong emotions and opinions. Depending on circumstances, elephants may be viewed as objects of worship, beasts of burden, food for a village, an endangered species worthy of the highest conservation efforts, or as crop-raiding killers.

The highly specialized morphology of the elephant is depicted by John Godfrey Saxes' (1817–1887) poem based on the famous Indian fable (see page xv). We chose to present this poem because much like the blind men, there is still much we do not know about elephants.

As long as humans have kept elephants in captivity, their health care has been a topic of concern. One of the earliest recorded treatments of an elephant was of "Kadol Etha" belonging to King Dutugemunu (161–137 B.C.). Kadol was treated for wounds sustained from molten metal. The first treatises on elephant health care were written in Asia over 2000 years ago (the Hastiayurveda and Gajasastra). Scientific articles began to appear in the 19th century. In the 20th century the works of G. H. Evans (*Elephants and Their Diseases,* 1910), A. J. W. Milroy (*Management of Elephants in Captivity,* 1922, republished by S. S. Bist in 2002), Francis Benedict (*The Physiology of the Elephant,* 1936), G. Pfaff (*Reports on the Investigation of Diseases of Elephants,* 1940), A. J. Ferrier (*The Care and Management of Elephants in Burma,* 1947), Sylvia Sikes (*The Natural History of the African Elephant,* 1971), U Toke Gale (*The Burmese Timber Elephant,* 1974), and others certainly contributed to our collective knowledge of elephant care and husbandry at the time.

But despite the fact that one-third of all Asian elephants remaining in the world are in captivity, no modern comprehensive text on elephant medicine and surgery exists. The editors and contributing authors hope that this volume will begin to fill that void. Thirty-six scientists and clinical veterinarians have shared their expertise and experiences to compile information on biology, husbandry, and veterinary medicine and surgery of the elephant as we know it today.

Each author presents his or her experiences plus those of others expressed in the literature. Although not an exhaustive literature review, over 3000 references are cited to provide readers the opportunity to delve more deeply into specific topics. The opinions expressed are those of the authors.

Free-ranging elephants face a precarious future. Habitat loss, poaching, and exploitation are decimating many populations to near extinction. Elephants and man compete for limited space and resources in Africa and Asia. Reports of human-elephant conflict appear in the news almost daily, with losses incurred on both sides.

Captive or "domesticated" elephants in Asia also face uncertainty because the timber industry in most Asian countries no longer requires the labor once provided by elephants. Many of these elephants now find themselves in an urban environment with no chance of foraging for natural feeds and often no access to proper veterinary care.

Anecdotal information has always been and will continue to be important to the care of elephants. It is hoped that this book will open a venue for the greater sharing of such information. At the same time the

paucity of information currently available on some topics may help to focus attention on areas of needed research. Those with special expertise and experience who have a bearing on the topics involved in the book are invited to contact the editors so that a future edition may reflect expanded information and other viewpoints.

Elephants deserve our care and our concern for their welfare.

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# The Blind Men and the Elephant

It was six men of Indostan
To learning much inclined,
Who went to see the Elephant
(Though all of them were blind),
That each by observation
Might satisfy his mind.

The First approached the Elephant,
And happening to fall
Against his broad and sturdy side,
At once began to bawl:
"God bless me! but the Elephant
Is very like a wall!"

The Second, feeling of the tusk
Cried, "Ho! what have we here,
So very round and smooth and sharp?
To me 'tis mighty clear
This wonder of an Elephant
Is very like a spear!"

The Third approached the animal,
And happening to take
The squirming trunk within his hands,
Thus boldly up he spake:
"I see," quoth he, "the Elephant
Is very like a snake!"

The Fourth reached out an eager hand,
And felt about the knee:
"What most this wondrous beast is like
Is mighty plain," quoth he;
"'Tis clear enough the Elephant
Is very like a tree!"

The Fifth, who chanced to touch the ear,
Said: "E'en the blindest man
Can tell what this resembles most;
Deny the fact who can,
This marvel of an Elephant
Is very like a fan!"

The Sixth no sooner had begun
About the beast to grope,
Then, seizing on the swinging tail
That fell within his scope.
"I see," quoth he, "the Elephant
Is very like a rope!"

And so these men of Indostan
Disputed loud and long,
Each in his own opinion
Exceeding stiff and strong,
Though each was partly in the right,
And all were in the wrong!

# BIOLOGY, MEDICINE, AND SURGERY OF ELEPHANTS

# 1 Taxonomy, Classification, History, and Evolution of Elephants

Jeheskel Shoshani

## INTRODUCTION

*Biological classification* is categorization and organization of organisms by their unique characters. A sound classification with standardized scientific names provides a universal language for laymen and scientists alike in cases where common names are not standardized.

During the 18th and 19th century, however, the concept of homology was just beginning to emerge, and grouping of animals was based on external morphology and habitat. In this system, manatee was grouped with seals (as "Aquatilia" of Scopolli 1777) or with cetaceans (as "Natalia" of Illiger 1811), and elephants were grouped with rhinoceroces and tapirs as "Pachydermes" (of G. Cuvier 1800). A summary of these earlier ideas of classfication is given in Shoshani.[35]

In modern times, to classify an organism, a researcher must follow certain rules and procedures. To facilitate classifications, taxonomists developed guidelines, the Code of Nomenclature (International Commission of Zoological Nomenclature 1999)[20] (referred to hereafter as "the Code"), which is updated on a regular basis. The Code guides the naming of the taxa, not their discovery and conception. It emerges that classification, taxonomy, systematics, and phylogeny are all interrelated. Information from one discipline can be applied to another; this can easily be understood when comparing their definitions (these terms are defined in the section "Definitions" below). The field of phylogeny, however, requires some elaboration. Relationships among taxa can be obtained and tested using cladistic or phylogenetic methods, employing morphological or molecular characters. In ideal situations, results from both approaches corroborate each other. One school of thought holds that cladistic or phylogenetic relationships should be reflected in the classification (more on that later).[16]

The main purpose of classifying animals and plants is to facilitate better communication among scientists. An example of the applicability of classification and also of phylogeny is in the fields of communicable diseases, zoonotic diseases, susceptibility and resistance to diseases in general (discussed later), and organ transplant. The more closely related two animals are, the more likely that incompatibility will be reduced and the better chances for a successful transplant.[42]

## DEFINITIONS

**Clade.** A cluster of taxa derived from a single common ancestor.

**Cladistic methods.** A mode of classification based, in principal, on grouping of taxa that possess shared, similar ("derived") characters that differ from the ancestral condition.

**Cladogram.** A tree diagram representing phylogenetic (or cladistic) relationships among taxa based on their shared-derived characters or synapomorphies.

**Classification.** The practice of grouping organisms into a hierarchy of categories ranging from subspecies, species, genera, families, orders, classes, phyla, and kingdoms (except for the subspecies, all these are obligatory categories, see definition below). Taxa included in each of these categories are entities to themselves encompassing unique features. Thus, organisms classified in a species are more similar to each other than they are to members of other species in the same genus. Similarly, genera in one family share more characters with each other than with genera in other families, and so on.

**Grade.** Distantly related or unrelated species (or taxa) that reach the same level due to parallel or convergent evolution.

**Homology.** Shared similarity due to common descent.

**Nomenclature.** The practice of giving names to animals and plants.

**Obligatory categories in classification.** The major ranks (or categories) that are usually employed in classification of organisms. They include the species, genus, order, family, class, phylum, and kingdom. All other categories, such as those with the prefix *sub-* or *super-* (e.g., subfamily, superfamily, subclass, and superclass) are not obligatory of formal, general classification.

**Phylogeny.** The evolutionary history of common descent or of a lineage (that is, of a species or a group of species) as related to their ancestor-descendant relationships. In a restricted sense, the history of descent of a given set of taxa.

**Species.** A basic taxonomic category. In the biological species concept, a species (taxon) includes interbreeding or potentially interbreeding populations possessing unique characters and reproductively isolated from other such groups (taxa), under natural conditions.

**Systematics.** The study of diversity of organisms and their comparative and evolutionary relationships (= classification and taxonomy).

**Taxon (plural taxa).** A group of organisms that share common characters, included at any level of the classifications (e.g., species, genus, or family).

**Taxonomy.** The discipline including the rules and procedures used to classify organisms.

## CLASSIFICATION IN HISTORICAL PERSPECTIVE

Among the earliest attempts to organize and classify organisms was that attributed to Aristotle, the Greek philosopher and naturalist (384–322 B.C.). Aristotle, it is believed, came to view nature as a continuum of organization, from lifeless matter through the complex forms of plants and animals. Carolus Linnaeus (Latinized name from Carolus Linné, lived from 1707–1778), a Swedish botanist, was the first authority to develop a formal classification scheme for organisms, giving them two-part names (hence the term *Binomial Classification*); the first is the genus and the second is the species, and both are descriptive names. This system is still used by most taxonomists. The 10th edition of Linnaeus's book *Systema Naturae*[23] (published in 1758) is considered the primary treatise on classification, and 1758 is taken to be the beginning date for which published scientific names are valid.

It is important to keep matters in perspective. Linnaeus was a devout, religious man. This was reflected in his belief that the number of species created was limited. In this context, the African and the Asian elephants belonged to one species. Thus, the name *Elephas maximus* given by Linnaeus in 1758 was apparently based on a fetus of an African elephant and a specimen of the Asian elephant. It is believed that *Elephas maximus* of Linnaeus combines these two different elephant species—*Elephas* for the Asian elephant, and *maximus* for the African elephant, the larger of the two species (details are given in Shoshani and Tassy,[40] pp. 354 and 360).

The etymology of the word *elephant* or *Elephas* is from *ele*, a Greek derivative meaning an arch, and *phant* or *phas* from the Greek/Latin derivative meaning *fantastic* or *huge*. Thus, *elephant* or *Elephas* translates into a huge arch (from the shape of an elephant in side view). A separate scientific name for the African elephant (*Loxodonta africana*) was coined in 1827, 69 years later. The genus name *Loxodonta* describes the lozenge pattern of the enamel loops on the chewing (occlusal) surface of the tooth, and the species name, *africana* (note lowercase *a*) refers to the origin and habitat of this animal; it is usually found in savannahs of sub-Saharan Africa. The other elephant species in Africa is the forest African elephant (*Loxodonta cyclotis*), found in forested regions of central and western Africa. The species name *cyclotis* describes the roundish shape (*cycl*) of the ear (*otis)*. In the *africana* species the ear has a trapezoidal shape. Not all authorities subscribe to the two species concept of the African elephant; some still hold that there is one species with two subspecies—*L. a. africana* and *L. a. cyclotis* (more on that below).

## LINNAEAN CLASSIFICATION AND THE CODE

The Binomial Classification, established by Linnaeus in 1758, is the most commonly used system today. The Code of Nomenclature is an attempt to standardize the work of taxonomists and systematists, including nomenclaturists and classifiers, and to provide some published guidelines and rules that are regularly updated (see International Commission of Zoological Nomenclature 1999).[20] Binomial Classification and the Code are closely related, but for practical purposes, I present the two subjects separately.

### Binomial and Trinomial Classification

Recall that in this system, each species is given two names: the genus and the species. Both names must be Latinized, although their origin may be Latin, Greek, or another language. The first letter of the genus is written in uppercase and all subsequent letters of the genus and species are in lowercase, even if the species name is after a locality or a person. For example, the scientific name of the Asian elephant is *Elephas maximus* and that of the American mastodon is *Mammut americanum* (to accentuate the names they are italicized, underlined, or written in different formats from the rest of the text). When there is sufficient anatomical evidence to divide a species into two or more subspecies, we use three names

(hence the term *Trinomial*). For example, the scientific name of the Asian elephant from Sri Lanka is *Elephas maximus maximus*; other examples are given below.

The next step in this process is to place the species in a higher category. In the Linnaean system of classification, the primary or obligatory categories, from higher to lower are Kingdom, Phylum, Class, Order, Family, Genus, and Species. It is not an easy matter to decide into which hierarchy or category to place a newly found species. The criteria that govern this decision have to do with the differences between genus and family level, and they are related to the size of the gap of characters between different categories. Suffice it to say that in the example of the Asian elephant given above, scientists determined that, based on anatomical characters, *Elephas maximus,* the African elephant (*Loxodonta africana*), and woolly mammoths (*Mammuthus primigenius*) are distinctly unique yet they share similar characters and should be grouped in the subfamily Elephantinae, family Elephantidae. The skeleton of *Mammut americanum* possesses very different sets of characters; thus, it was decided to classify it in another family, the Mammutidae. Elephantidae, Mammutidae, and other families that share similar characters due to common ancestry were then grouped under the umbrella of a higher category, called Order, the Proboscidea. Related orders are classified in one Class, classes are grouped under a Phylum, and phyla under a Kingdom. An example of a simplified classification of the Proboscidea within Animalia is given in Table 1.1. Note that the suffixes of

**Table 1.1.** A Partial, Simplified Classification of Proboscidean Taxa[*]

| Category (= Rank) | Taxon |
|---|---|
| Kingdom | Animalia |
| Phylum | Chordata |
| Subphylum | Vertebrata |
| Class | Mammalia |
| Nonranked | Uranotheria (= Paenungulata)[a] |
| Order | Hyracoidea |
| Nonranked | Tethytheria |
| Order | Sirenia |
| Order | Proboscidea |
| Nonranked | Mammutida[b] |
| Superfamily | Mammutoidea[b] |
| Family | Mammutidae[b] |
| Genus | *Mammut*[b] |
| Species | *Mammut americanum*[b,c] |
| Nonranked | Elephantida |
| Superfamily | Gomphotherioidea[b] |
| Family | Gomphotheriidae[b] |
| Genus & Species | *Gomphotherium angustidens*[b] |
| Superfamily | Elephantoidea |
| Family | Stegodontidae[b] |
| Genus & Species | *Stegodon zdanskyi*[b] |
| Family | Elephantidae |
| Subfamily | Elephantinae |
| Tribe | Loxodontini |
| Genus & Species | *Loxodonta cyclotis*[d] |
| | *Loxodonta africana*[e] |
| Tribe | Elephantini |
| Genus & Species | *Elephas maximus*[f] |
| Subspecies | *Elephas maximus sumatranus*[g] |
| | *Elephas maximus indicus*[h] |
| | *Elephas maximus maximus*[i] |
| Genus & Species | *Mammuthus primigenius*[b,j] |

[*]Refer to Figure 1.1 for depiction of the species on the cladogram; modified after Shoshani 2000, p. 22, and other sources.
[a]After McKenna et. al. 1997.
[b]= extinct.
[c]The American mastodon, now extinct, osteological remains were found in North America.
[d]The Forest African elephant, living (see text for possible use of the subspecies name *Loxodonta africana cyclotis*).
[e]The Bush or Savanna African elephant, living (see text for possible use of the subspecies name *Loxodonta africana africana*).
[f]The Asian elephant, living.
[g]The Sumatran Asian elephant subspecies, living (found on the island of Sumatra).
[h]The Indian, or mainland Asian elephant subspecies, living (found in India and Indochina).
[i]The Sri Lankan Asian elephant subspecies, living (found on the island of Sri Lanka, formerly Ceylon).
[j]The woolly mammoth, extinct; remains and intact carcasses were found frozen in the Arctic. The Colombian mammoth (*Mammuthus columbi*), extinct; remains were found in North America.

family names in any classification of animals are always *idae* and those of subfamilies are *inae*. These two conventional suffixes help identify quickly these categories or ranks.

In this classification (Table 1.1), it is noted that entries are indented such that the taxon listed below is nested within the taxon listed above it. This system embodies the idea that one or more species are grouped in a genus, one or more genera are grouped in a family, and so on. Some aspects of the process of giving names of ranks to certain taxa are discussed below.

Table 1.1 also reflects the relationships among Hyracoidea (hyraxes), Sirenia (manatees and dugongs), and Proboscidea.[26] Shared, derived, characters among Hyracoidea, Sirenia, and Proboscidea include serial versus alternate carpal bones, and affinity between Sirenia and Proboscidea include bifid heart[39] (see also figure on p. 16 of Shoshani).[37]

### The Code

Being a reference of standard terminologies, recommendations, and rules, the Code is the authority for a taxonomist. An important rule in nomenclature and classification is the Principle of Priority (published in the Code, see International Commission of Zoological Nomenclature 1999).[20] This principle states that if two different names have been given to the same animal or plant by two different researchers, the one that was published first is valid. For example, in 1817 the famous French anatomist Georges Cuvier coined the name *Mastodonte* for an animal that was found in Big Bone Lick site, not far from the Ohio River, Kentucky, USA. It appears that G. Cuvier and C. S. Rafinesque (who in 1814 coined the name *Mastodon* for the same animal) were not aware of the publication of Johan F. Blumenbach, a German naturalist, who, in 1799, named the same animal *Mammut*. Following the Principle of Priority, the older name has prevailed (details in Shoshani and Tassy,[40] p. 351).

If only the genus name is employed, the author of the name and the year it was published follows it—e.g., *Mammut* Blumenbach, 1799. If, however, the species name is also to be included, it also is followed by the author and year of publication—*Mammut americanum* (Kerr, 1792). Note that the author and year are written inside the parentheses ( ). This is because the original Type Species name coined by Kerr for the same animal was *Elephas americanus*; thus, the credit still goes to the original author who first named the species, even though it is no longer a valid original genus name (in this case, *Mammut* is the valid generic name, see above).

Another important rule in the Code is the Latinization of scientific names. In 1825, F. Cuvier coined the name *Loxodonte* for the African elephant. This name is not valid because it is not Latinized (Article 11[b] of the Code). In 1827 the Latinized version of this name (*Loxodonta*) was used in the journal where the review of F. Cuvier's work appeared. It was not clear from the text who was the writer who Latinized the name; for this reason, the scientific name of the African elephant appears as *Loxodonta* Anonymous, 1827[40] (details in Shoshani and Tassy 1996, p. 361).

## NUMBERS OF PROBOSCIDEAN SPECIES AND SUBSPECIES

### Living and Extinct Taxa

In 1942 Henry Fairfield Osborn recognized 352 species and subspecies of Proboscidea, living and extinct. The most recent revision was that of Shoshani and Tassy,[40] where they recognized 177 species and subspecies classified in 43 genera and at least 10 families. Since then Sanders[34] named one new genus and five new species, bringing the total to 182 species and subspecies and 44 genera. Of these, today there are two extant genera, with three species. Living elephants are listed by CITES (Convention on International Trade in Endangered Species of Wild Fauna and Flora) either in Appendix I or in Appendix II. Appendix I includes taxa that are threatened with extinction and are or may be affected by trade. Appendix II species need not be threatened, but either require regulation so that they do not become so or must be listed to help control trade in other species, the so-called "look-alike species."[30] The vast majority of living elephant populations are continuously decreasing due to shrinking range or habitat fragmentation.

### Living Taxa

**Generalized Features and Medicine.** Today we recognize three living species of elephants, classified in two genera—*Loxodonta* and *Elephas* (Tables 1.2 and 1.3 include differences between these genera). Based on the available morphological evidence, *Loxodonta*, represented by the living African elephant, appears to be more primitive than *Elephas,* represented by the living Asian elephants. Both *Loxodonta* and *Elephas* originated in East Africa, and yet *Loxodonta* is believed to embody more generalized features than *Elephas.*[25,40] From the medical standpoint, it is noted that generalized mammals (e.g., insectivores) are better adapted than specialized mammals (e.g., horses) to cope with living in different habitats.[3,4] It could be argued that generalized mammals may be better adapted to fight diseases than specialized mammals. To test this hypothesis a survey of diseases known to occur in the African versus the Asian elephant should be conducted. It is predicted that the African species (*L. cyclotis* and *L. africana*) would be more resistant to diseases—including communicable and zoonotic diseases—than the Asian species (*E. maximus*). Recent investigators have demonstrated different susceptibilities to herpesvirus infection in captive Asian versus African elephants[32] and the apparent increased prevalence of uterine cystic endometrial hyperplasia[2] and uterine leiomyomas[17] in Asian elephants. These

**Table 1.2.** Major Differences Between the African and the Asian Elephant

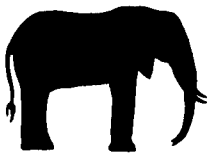
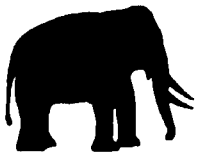
| |  |  |
|---|---|---|
| Weight | 4,000-7,000 kilograms (8,820-15,430 pounds) | 2,000-5,500 kilograms (4,410-11,020 pounds) |
| Height at shoulder | 3-4 meters (10-13 feet) | 2-3.5 meters (6 feet 7 inches-11 feet 6 inches) |
| Skin | More Wrinkled | Smoother |
| Number of ribs | Up to 21 pairs | Up to 20 pairs |
| Highest point | At top of shoulder | At top of head |
| Size of ears (pinnae) | Larger, do exceed height of neck | Smaller, do not exceed height of neck |
| In mature individuals | | |
| dorsal of pinnae | Fold medially | Fold laterally |
| Shape of back | Concave | Convex or level |
| Shape of belly | Slopes diagonally downwards from front to back | Either almost horizontal or "sagging " in the middle |
| Shape of head | No compression; no bulges, no dish | Compressed antero-posteriorly; has dorsal bulges, dished forehead |
| |  |  |
| Teeth | Lozenge-shaped loops | Narrow compressed loops |
| |  |  |
| Food | Mostly browser | Mostly grazer |
| Tusks | Both sexes possess tusks; larger in males | Males usually carry tusks; in females tusks are vestigial or absent |
| Trunk | Has more rings (annulated), less rigid | Appears to have less annulation, more rigid |
| Tip of trunk | Two "fingers" | One "finger" |
| |  |  |
| Number of nail-like structures (toes) | Forefeet 4 or 5<br>Hind feet 3, 4 or 5 | Forefeet 5<br>Hind feet 4 or 5 |

**Table 1.3.** Major Differences Among Species and Subspecies of Elephants

Within the African Elephants, *Loxodonta* sp.*

| | Bush Species (*L. africana*) | Forest Species (*L. cyclotis*) |
|---|---|---|
| Weight | 4,000–7,000 kilograms (8,820–15,430 pounds) | 2,000–4,500 kilograms (4,410–10,000 pounds) |
| Height at shoulder | 3–4 meters (10–13 feet) | 2–3 meters (6 feet 7 inches–10 feet) |
| Skin | On average lighter | On average darker |
| Shape and size of ears | Triangular, extend below line of neck | Rounder, do not extend below line of neck |
| Skull, cranium | Much pneumatized | Less pneumatized |
| Skull, mandible | Shorter | Longer |
| Tusks | Curved out and forward, thicker | Straighter, down-pointing, slender |
| Number of naillike structures ("toes") in adults | Forefeet 4 or 5<br>Hindfeet 3, 4 or 5 | Forefeet 5<br>Hindfeet 4 or 5 |

Within the Asian Elephants, *Elephas maximus***

| | Sri Lankan Subspecies (*E. m. maximus*) | Mainland Subspecies (*E. m. indicus*) | Sumatran Subspecies (*E. m. sumatranus*) |
|---|---|---|---|
| Weight | 2,000–5,500 kilograms (4,410–12,125 pounds) | 2,000–5,000 kilograms (4,410–11,020 pounds) | 2,000–4,000 kilograms (4,410–8,820 pounds) |
| Shoulder height | 2–3.5 meters (6 feet 7 inches–11 feet 6 inches) | 2–3.5 meters (6 feet 7 inches–11 feet 6 inches) | 2-3.2 meters (6 feet 7 inches–10 feet 6 inches) |
| Skin color | Darkest, with large and distinct patches of depigmentation on ears, face, trunk, and belly | Color and depigmentation in between the other two subspecies | Lightest with least depigmentation |
| Size of ears | Most have large ears | Vary in size | Appear large compared to body size |
| Tusks incidence | Lowest | Intermediate | Possibly the highest |
| Number of ribs | 19 pairs | 19 pairs | 20 pairs |

**Loxodonta cyclotis* is more primitive than *L. africana* for these reasons: forest dweller, smaller, slender, and down-pointing tusks and other skull characters discussed by Grubb 2000.

***Elephas maximus sumatranus* is possibly the most primitive Asian subspecies for these reasons: forest dweller, smallest, has largest number of ribs, possibly has highest incidence of tusks, has least depigmented skin and other characters discussed by Deraniyagala 1955.

studies, however, examined only captive individuals in which husbandry and management issues may confound any genetic or taxonomic effect. Further research in this area would be valuable in the practical management of elephant populations as well as enhancing our general understanding of the association between taxonomy and the balance between health and disease.

Taxonomy's importance in understanding the potential health problems of elephants is highlighted by the work of Hagey[15] who described the unique use of bile alcohols in elephants and a few of their closest relatives, the manatee and hyrax. All other mammals produce bile acids as a product of cholesterol metabolism. The presence of bile alcohols instead of acids may make elephants more susceptible to bacterial invasion and cholelith formation.[1]

An understanding of taxonomy is also valuable when considering potential metabolic and physiologic similarities in drug metabolism.[21,29] Similar digestive tracts or similar cholesterol metabolic pathways, for example, might be empirically expected to absorb or process a particular drug similarly, allowing veterinarians to extrapolate drug doses from one species to another. Again, little research into the comparative pharmacology of nondomestic animals has been done for any species, including elephants. See Chapter 15 for further information.

**The *Loxodonta* Group.** Traditionally, the African elephant was divided into two subspecies: *L. africana africana* (the bush African elephant) and *L. a. cyclotis* (the forest African elephant, discussion in Grubb[14] 2000). Recent taxonomic revision within this group is manifested in dividing the African elephant into two species: the forest African elephant (*L. cyclotis*), and the bush African elephant (*L. africana;* see Table 1.3, upper). This taxonomy is not agreed upon by all scientists. Grubb and Roca support the species concept,[14,33] whereas Debruyne[5] provides data in support of the traditional subspecies, *L. a. africana* and *L. a. cyclotis*. Between the two African species, *L. cyclotis* is more primitive than *L. africana* (discussed in detail by Grubb; see also footnotes to Table 1.3). In addition to the two species of African elephants, Eggert reported on what

might be interpreted as a possible third species of African elephant for the populations of the forest and savannah elephants of West Africa (these interpretations are not widely accepted).[7] These findings are based on DNA extracted from dung of elephants in Ghana, the Ivory Coast, Mali, and Cameroon. These elephants live in both forest and savannah habitats. The study suggests that, based on genetic data, the West African populations have been isolated from other elephant populations for as long as 2.4 million years.

**The *Elephas* Group.** We find less controversy in the taxonomy of the Asian elephant (*E. maximus*), where Shoshani and Eisenberg (1982) recognized three subspecies: the Sumatran Asian elephant (*E. m. sumatranus*), the mainland Asian elephant (*E. m. indicus*), and the Sri Lankan Asian elephant (*E. m. maximus*).[38] Evolutionary trend among these subspecies is suggested. Thus, *E. m. sumatranus* is said to be the most primitive of the three subspecies, *E. m. maximus* the most derived, and *E. m. indicus* an intermediate form. Evidence for this trend includes 20 pairs of ribs in *E. m. sumatranus* and 19 pairs in *E. m. maximus* and *E. m. indicus*.[37,43] Other features include forested versus less-forested dwelling; small versus large body size; ear size; possibly high versus low incidence of tusks, tusk size, and shape (e.g., straight versus curved); and least versus most skin depigmentation. Additional characters and discussion on Asian elephant subspecies were provided by Deraniyagala.[6] Table 1.3 summarizes these differences betweeen the subspecies of the Asian elephant.

A recent study by Fernando[8] concluded, based on DNA isolated from dung, that the elephants from Borneo island (specifically the Malaysian states of Sabah and Sarawak) are ". . . genetically distinct, with molecular divergence indicative of a Pleistocene colonization of Borneo and subsequent isolation." These authors suggest ". . . that a formal reinstatement of the *E. m. borneensis* taxa await a detailed morphological analysis of Borneo elephants and their comparison with other populations." This author concurs with Fernando[8] that there should also be morphological differences among the recognized Asian elephant subspecies. Additionally, it would also be a stronger argument for the proposed subspecies if the recent findings of Fernando would be repeated and corroborated.[8]

## CAN THE TWO LIVING SPECIES INTERBREED?

Traditionally, a species was defined as a group of animals (taxon) that possesses unique characters and does not interbreed with other such groups (taxa) under natural conditions.[27] Since then it has been observed that hybrid zones between distinct species in the wild have been reported for warm-blooded vertebrates, both birds[28] and mammals[13]. In captivity, however, the animals are artificially placed together, and hybrids among animals that will never meet in the wild may occur. Such was the case of "Motty," the only known hybrid between a male African elephant, "Jumbolino" ("Bubbles"), and a female Asian elephant, "Sheba." This hybrid was conceived in Chester Zoo, England, in 1978.[18] Motty lived only 10 days; his skin is mounted at the Natural History Museum (formerly British Museum of Natural History, London). Zoo authorities and other people doubted whether it would have been possible for the two elephant genera to hybridize. Unfortunately no soft tissue samples were kept for future testing, but a sample of small dry skin was collected from behind the ear of Motty and was used in immunological experiments to test whether Motty was indeed a hybrid. The results confirmed that Motty's tissue behaved like that of a mule, corroborating that it was a hybrid between *Loxodonta africana* and *Elephas maximus*.[24] These results are not totally surprising because the diploid chromosomes number in somatic cells for both elephant species is 56.[19]

## A SIMPLIFIED CLADOGRAM OF SELECTED PROBOSCIDEANS

Evolutionary relationship can be depicted either as a "family tree" or as a cladogram (Fig. 1.1). A family tree may be compared to a genealogical family tree where the origins of the great-great-grandparents of an individual are being traced. In a family tree, such as the one given in Shoshani[37] (pp. 26–27), the direct line of ancestry of *Elephas* (the animal at the center, top) is drawn as passing through *Primelephas*, *Gomphotherium*, *Palaeomastodon,* and "Ancestral proboscideans." All other proboscideans depicted are side branches and are not a part of the main tree trunk. In this kind of illustration the main trunk is conceived as an evolutionary grade including taxa that are not necessarily phylogenetically related. In a cladogram, taxa are depicted successively as sister taxa, and the common ancestors are reconstructed, not observed, presumed at the point of convergence of two sister taxa. For example, the common ancestor of *Mammuthus* and *Elephas* in Figure 1.1 is presumed at a point just above the tribe name Elephantini. The branch of the sister taxa Elephantini and Loxodontini are joined to form the subfamily Elephantinae. The common ancestor of Elephantini and Loxodontini may have been an animal that embodied characters similar to those of *Primelephas*.

To better understand this cladogram, examine Figure 1.1 in tandem with the classification provided in Table 1.1. In this table taxa are listed in the sequence as they would appear on a cladogram from the most primitive or generalized (listed first) to the most derived or specialized (listed last). This is a simplified cladogram with a simplified table depicting only a portion of the

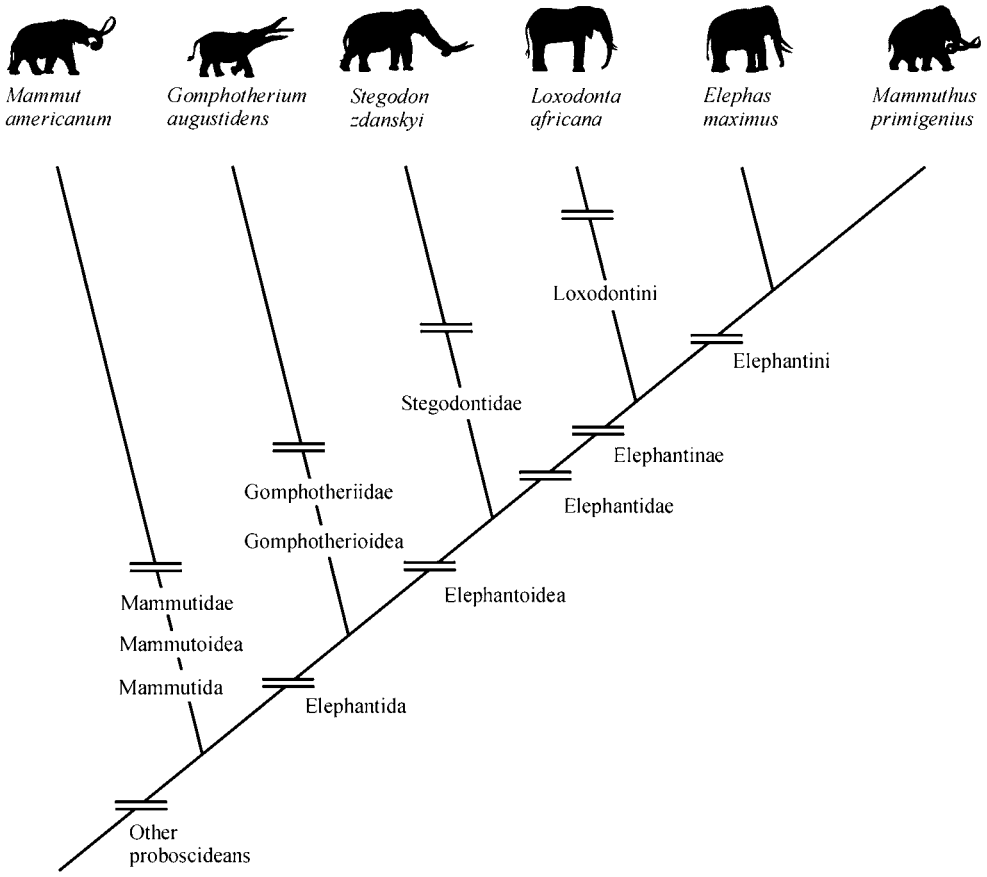



***Figure 1.1.*** *A cladogram of selected proboscideans (modified after Shoshani and Tassy 2005, p. 14), to be studied in tandem with Table 1.1. Reprinted from Quaternary International, Volume 126-128, J. Shoshani and P. Tassy, Advances in Proboscideans Taxonomy & Classification, Anatomy & Physiology, and Ecology & Behavior, page 14, copyright (2005), with permission from Elsevier.*

Proboscidea. When all the 44 genera of Proboscidea are included, the branching pattern of the cladogram is no longer simple. It would be even more complicated if we include all 182 species and subspecies.

## EVOLUTIONARY TRENDS AND MIGRATION OF PROBOSCIDEANS

### Evolutionary Trends

As we proceed from the earliest proboscidean that lived in early Eocene epoch (about 55 million years ago) to the present (Holocene), we observe these major evolutionary changes or trends: overall increase in body size; increase of tusk size; development of a trunk, or proboscis; and increase of trunk length (these trends are depicted in Shoshani,[37], pp. 26–27). Table 1.4 elaborates on these trends and includes information on gigantism (over 4 meters shoulder height) and dwarfism (only 1 meter tall), coevolution of infrasonic communication and the ability to store water in the pharynx, and horizontal displacement of premolars and molars as though they were moving on a slow conveyor belt (Fig. 1.2). *Phosphatherium,* the earliest known proboscidean, was about the size of a dog (10–15 kg), but it was not a dwarf; it did not have a trunk, tusks, or horizontal displacement of premolars and molars (these features developed later within the Proboscidea). Nevertheless, *Phosphatherium* was a proboscidean since it possessed unique proboscidean characters such as a well-developed zygomatic process of the maxillary bone.[12]

### Migration of Proboscideans

A map of migratory routes, as those depicted in Figure 1.3, was constructed based on fossil material discovered at different localities, at different geological times. Thus, the older the fossils, the closer they would appear to the place of origin of the Proboscidea. For example, numidotheres (e.g., *Phosphatherium,* the earliest known proboscidean; *Daouitherium*; and *Numidotherium*) were found in Morocco and Algeria, northwest Africa, in the early-middle Eocene. Africa is believed to have been isolated from other continents during most of the Paleogene (Paleocene, Eocene, and Oligocene), and thus its fauna during these geological epochs was endemic. We are uncertain of the exact origin of Proboscidea. Emmanuel Gheerbrant (personal communication, 2005) suggested: "Paleogene proboscideans are representative of the whole African province, proboscideans are of African origin" (see also [9,10]). For this reason, migration and dispersal patterns of the earliest proboscideans from northwest Africa during the Paleogene are uncertain (thus the question marks on the map). However, one possibility emerges that the northern shores of the Mediterranean Sea (a remnant of the an-

**Table 1.4.** Proboscideans Evolutionary Trends*

**Increase in size—**Earliest proboscideans were about the size of a dog; later taxa became giants, reaching over 4 meters at the shoulders. Dwarfism is observed in certain lineages, perhaps due to isolation (such as, but not limited to, islands); some were only 1 meter tall.

**Lengthening of limb bones and development of short, broad feet**

**Growth of the skull to extraordinarily large size—**This is particularly noticeable in the cranium, where greater surface for muscle attachment was possible. Enlargement of the cranium was facilitated by development of air cells (pneumatized bones), a feature that provides strength without added weight. Another possible function for the development of air cells is the need to protect the sensitive brain tissues from extreme environmental temperatures. The external surface of an elephant cranium can be about 25 cm from the brain; this physical protection with "padding" of air is probably a very important feature in the survival of certain proboscidean lineages.

**Coevolution of infrasonic communication and the ability to store water in the pharynx—**These developments appear to be associated with cranial and otic changes, modified hyoid apparatus, and evolution of the proboscis.

**Shortening of the neck—**The skull and its associated structures (tusks and trunk) became large and heavy and the neck was reduced, probably as a mechanical advantage for leverage.

**Elongation of the lower jaw (mandible) and secondary shortening of the cranium and mandible was an early primary trait among proboscideans—**Secondary shortening of the lower jaw (especially the area of the mandibular symphysis) and shift in the center of gravity of the head posteriorly was a trend associated with parallel evolution in advanced proboscideans.

**Development of a proboscis—**This observation is based on the elevated position of the external naris, enlargement of the infraorbital canal, the connection between frontal and premaxilla bones, and the shapes and sizes of the premaxilla and nasal bones. It is believed that the combination and elongation of the upper lip and nose have evolved to accommodate the distancing of the head from the ground due to the increase in size of the animal. Subsequently, the proboscis is further elongated to form a very mobile trunk, possibly having evolved independently in different lineages.

**Forward or horizontal displacement of cheek teeth (premolars and molars)—**The movement of teeth may be regarded as though they were moving on a slow conveyor belt; the earlier teeth are smaller than later ones. This feature is present in all known Neogene (Miocene through Pliocene epochs) proboscideans, from mammutid through elephantid species. The vast majority of other mammals, humans included, has vertical rather than horizontal tooth displacement.

**Reduction in number of teeth from the full eutherian dentition—incisors 3/3, canines 1/1, premolars 4/4, molars 3/3—**Throughout the history of the Proboscidea, there is a decrease in the numbers of premolars, canines, and incisors. Living elephants have this dental formula: 1/0 0/0 3/3 3/3.

**Hypertrophy (excess growth) of the middle incisors to form tusks—**Some of these were straight, curved downward, or upward and helicoidal; they functioned in food gathering, defense, offense, and display. Enamel covering of tusks decreased to a longitudinal lateral band and then disappeared. Tusks greatly increased in length and diameter; those of proboscideans are the largest known teeth of animals, living or extinct.

**In a cross section, tusks of advanced proboscideans (from members of Mammutidae to *Mammuthus*) exhibit Schreger pattern, also known as "engine turning" or guillochage—**In this system two sets of lines begin at the center and curve clockwise and counterclockwise toward the periphery; at the point of crisscrossing each other they form small rhomboid-shaped areas visible with the naked eye. This pattern is also present in dentine of the cheek teeth.

**Enlargement and specialization of the cheek teeth in proboscideans were achieved by increasing the number of cusps such as central conules, conelets, and the numbers of cross-lophs, or lamellae (from the simple 2 transverse lophs in the earliest members to 30 lophs in the most advanced taxa; large teeth of living elephants may weigh over 5 kg)—**This trend was accompanied by molarizing the deciduous premolars and thinning of enamel; it began in the early stages of proboscidean evolution. Parallel evolution, in the increasing number of lamellae, is found among the three genera of Elephantinae (*Loxodonta, Elephas, and Mammuthus*), and in *Stegodon*.

**Rate of evolution in the head, particularly the cheek teeth, has been faster than the rate of evolution of other organ systems in the body, e.g., the digestive system, which is relatively primitive and lags behind dentition.**

**Increase in the value Encephalization Quotient (EQ)—**One of the earliest proboscideans, the *Moeritherium* had an EQ of 0.2. This value increased during the 35–40 million of years and reached the value of up to 2.66 in modern elephantids.

*Slightly modified after Shoshani 1998, p. 484.

cient Tethys Sea) might be postulated as the place of origin of Proboscidea (for this discussion, members of Anthracobunidae are excluded). Northeast Africa (Egypt, Libya) embodied environmental conditions where fossils of *Moeritherium, Barytherium, Palaeomastodon, and Phiomia* were found in the late Eocene to Oligocene sediments. It seems plausible that northeastern African proboscideans may have migrated to the Horn of Africa (late Oligocene) and to East Africa (Miocene) where centers of radiation of some proboscideans (including deinotheres and gomphotheres) are believed to have taken place. Another center of radiation of extinct gomphotheres is believed to have occurred in Asia (a silhouette of *Gomphotherium angustidens* appears in Figure 1.1; details in Shoshani and Tassy).[40] From the Horn of Africa (again following the geological evidence we have thus far), it is suggested that some proboscideans (possibly gomphothere stock) migrated to what is today the Saudi Arabian peninsula (late Oligocene to early Miocene) and from there toward the general area of what is today Pakistan. Like the classification and the evolutionary tree (and cladogram), this map is subject to constant changes with the discovery of new fossils and/or different interpretations of old material. From this map of migration routes, we learn that Proboscidea was distributed in all the continents except Australia,

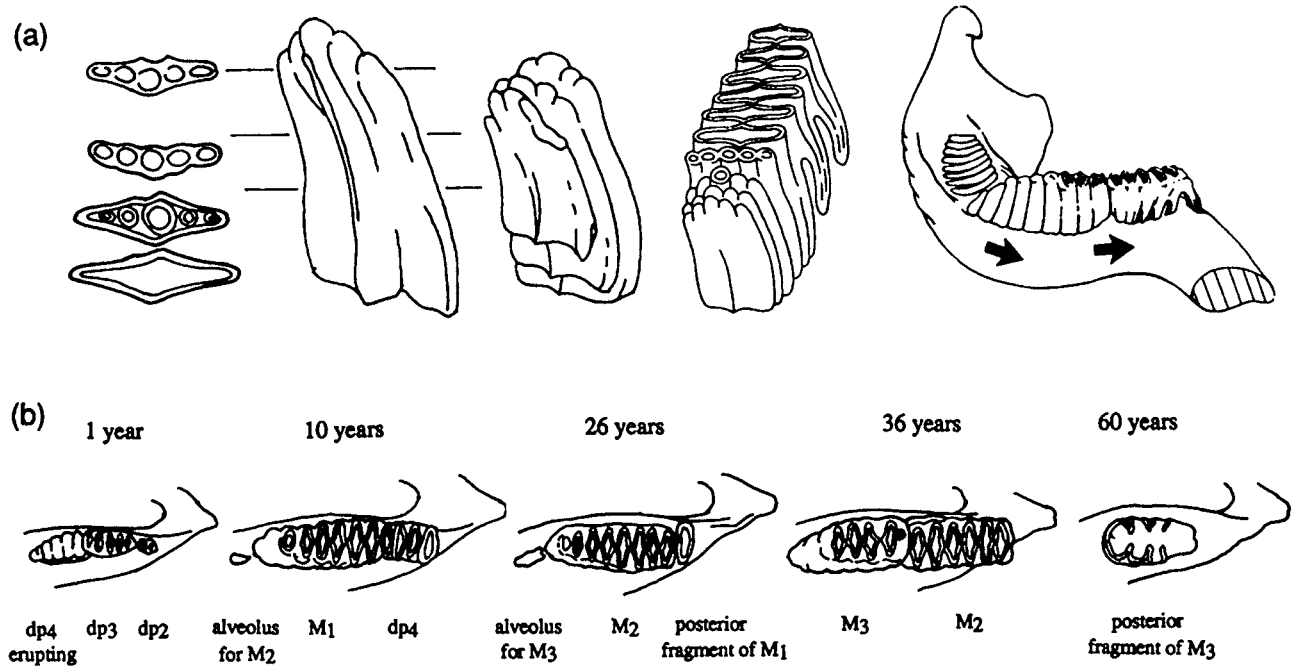



***Figure 1.2.*** *Diagrams depicting (a) cross-sections of isolated lamellae to reveal pattern of occlusal (chewing) surfaces, a tooth, and a left dentary in a medial view with arrows indicating direction of horizontal tooth displacement; (b) right sides of mandibulae of* Loxodonta africana *depicting teeth that are present at different ages (a, drawn from specimens by Gary H. Marchant; b, after Laws 1966, after Shoshani and Tassy 1996, p. 13).*

Antarctica, and some oceanic islands. Also included on this map are locations of pygmy proboscideans and a comparison of a typical elephant to a pygmy individual.

## CONCLUDING REMARKS

Biological classification involves categorization of organisms by their unique characters; it provides a universal language for laymen and scientists. Classification, cladograms, distribution maps, and suggested migratory routes will change as we discover new fossils or form different interpretations of previous data. Of the approximately 180 species and subspecies of proboscideans that inhabited the earth since early Eocene (55 million years ago) only two or three remain today and even these are in peril. We only begin to understand the possible relationships between taxonomy and medicine; it is plausible to hypothesize that the more generalized mammals may be better adapted to resist diseases than specialized mammals. Among elephants there is some evidence from captive animals that the African elephant (the more generalized or primitive species relative to the Asian elephant) is less susceptible to herpesvirus infection, uterine cystic endometrial hyperplasia, and uterine leiomyomas. Understanding taxonomy may help us better recognize the potential health problems of elephants.

## ACKNOWLEDGMENTS

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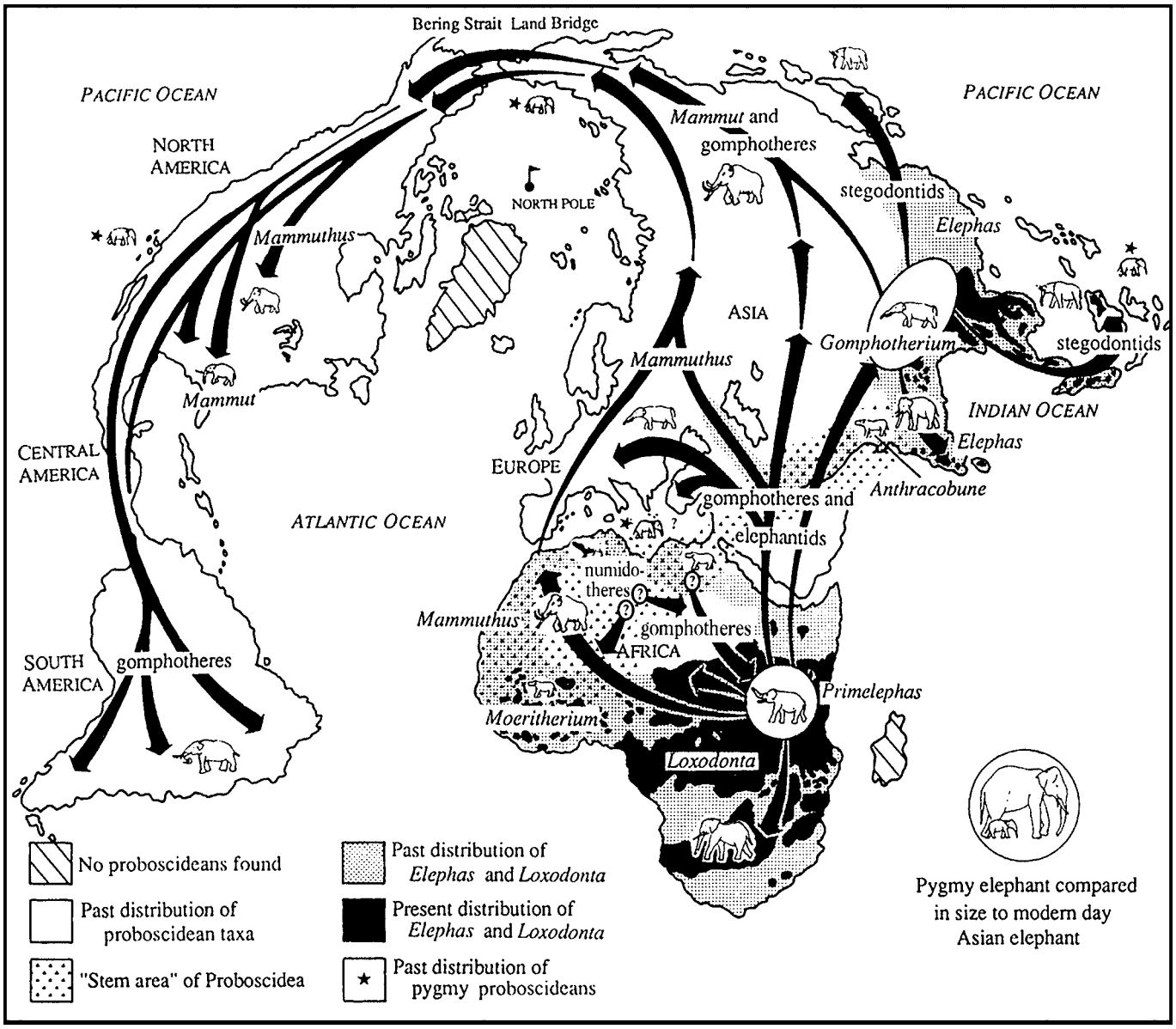


***Figure 1.3.*** *Suggested migratory routes of proboscideans (slightly modified after Shoshani and Tassy 1996, p. 341; Miniature* Phosphatherium, *after Gheerbrant and Bardet 1999).*


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# 2 Elephants in Captivity

Blair Csuti

## DOMESTICATED OR CAPTIVE ELEPHANTS?

"Nothing is more easy than to tame an animal, and few things more difficult than to get it to breed freely under confinement . . ." (Charles Darwin, 1859).

Elephants and their recently extinct relatives (mammoths, mastodons) were extensively hunted by stone-age humans. They also appear as frequent subjects in art dating to about 30,000 B.C. in Europe and Neolithic paintings throughout Africa.[5,18]

The domestication of many of today's common species of livestock dates to about 10,000 B.C.,[23] but the earliest evidence for captive elephants comes from images on soapstone seals left by the Harappan culture of the Indus Valley of present-day Pakistan, which flourished about 4,500 years ago. The first training of captive elephants likely predates the archaeological evidence by some time. Despite the long and complex relationship between humans and elephants, nearly all elephants ever brought into the service of ancient or modern civilizations have been captured from wild populations.

Lair[10] reviews the appropriateness of terms used to describe elephants captured and trained by humans. He reluctantly adopts the term *domesticated elephants* to refer to animals in the extant captive population largely because it is inclusive enough to accommodate individuals born in captivity, even if sired by wild males. Others[5,9] restrict the term *domestication* to plants or animals subjected to generations of selective breeding aimed at modifying behavioral or physical characteristics. Recognizing the general agreement that elephants in human service remain trained wild animals regardless of the circumstances of their birth, the term *captive elephants* most accurately describes the past and present status of their relationship with humans.

## HUMAN USE OF CAPTIVE ELEPHANTS

Being the largest and most powerful terrestrial vertebrate, elephants have evoked wonder, envy, and fear throughout recorded history. They have been worshiped, brutalized, and conscripted by both ancient and modern civilizations. Human uses of elephants span thousands of years. See Figure 2.1. Although wild individuals of all three species have been trained for captive use, it is surprising that, until recently, the savannah elephant of sub-Saharan Africa (*Loxodonta africana africana*) was never captured or trained for work.[5]

The following sections address broad categories of elephant use and abuse, reported by time and place. Although overlap in the roles elephants played in many historical milieus is unavoidable, focusing on their primary function offers the clearest view of their historical and practical interactions with human society.

### War Elephants

**Vedic India.** The Harappan civilization of the Indus Valley was replaced by Aryan invaders by 1500 B.C. The Aryans arrived on horseback but soon absorbed the elephant culture already present in India. Their epic texts, the *Ramayana* and the *Mahabharata*, describe the use of elephants in battle in the period 1000–700 B.C.[23] The first record of an elephant killed in battle dates to 1100 B.C.[27] Sukumar[23] cites Greek sources reporting that the Nandas (successors to the kingdom of Magadha) maintained an army of 3,000 elephants in the 4th century B.C. Although many early accounts of war elephant numbers may be overstated, they were clearly an integral part of Hindu armies of the first millennium B.C.

**Hellenistic Empire.** Ancient Greeks had reports of Indian elephants being successfully used in the defeat of Cyrus of Persia by Amoraius, king of the Derbikes, in 530 B.C.[23] Descriptions of the exploits of elephants in war become more detailed beginning with the encounter between the Macedonian army of Alexander the Great and Darius III of Persia at the battle of Gaugamela in 331 B.C. Darius brought 15 elephants to the battle, but lost both the day and his elephants to Alexander, who went

***Figure 2.1.*** *One of many war elephants sculpted at Angkor Wat, Cambodia, depicting battles between the ancient Khmer (led by their king, Suryavarman II, the builder of Angkor Wat) and their enemies.*[16] *(Image courtesy of Hank Hammatt.)*

on to accumulate over 100 more elephants in his push toward India. His trek ended in a victory over King Porus at the river Hydaspes (in the north of present-day Pakistan) in 326 B.C. Based on his experience in Persia, Alexander developed tactics that allowed his infantry to neutralize the force of as many as 200 elephants fielded by Porus, mainly by attacking the trunk and ankles. Because his cavalry was inexperienced in working with elephants, Alexander joined battle without his own elephant force. His superior strategy saved the day, but his troops were reluctant to pursue further conquests in India (possibly intimidated by rumors of thousands of war elephants awaiting them) and began a long trek back to the Mediterranean, taking some 200 elephants with their retreating column.[5] Alexander died in Babylon in 323 B.C., and the many claimants to his throne divided his kingdom. This led to 3 centuries of warfare in the Mediterranean and Near East in which both African and Asian elephants played spectacular and sometimes decisive roles.

The fragmentation of Alexander's empire left the Seleucid Kingdom in control of the Middle East and Persia. Seleucus I came to terms with Chandragupta Maurya, who gained control of the Nanda throne in 321 B.C., reputedly with an army that included 9,000 elephants.[23] The Seleucids received 500 war elephants, which were to prove decisive in a battle with another of Alexander's successors, Antigonus, at Ipsus in 301 B.C. Although Antigonus fielded 70 of his own war elephants, his forces were routed by Seleucid elephants, leading to his defeat and death.[5] The Sassanid dynasty (A.D. 224–651) evolved into the Second Persian Empire. Persian King Saphur II used his war elephants as late as 311 A.D. while crushing a Christian rebellion at Susa.[5]

Pyrrhus, king of the Greek city-state of Epirus, introduced Rome to Asian war elephants when he invaded Italy in 280 B.C. He arrived at Tarentum, the largest Greek city-state in southern Italy, with 25,000 men and 20 war elephants.[5] His elephants were decisive in many Roman defeats, though his losses were so great at Asculum that such a triumph is still called a "Pyrrhic victory." While winning further battles on the mainland of Italy and in Sicily in years following, he was eventually defeated by Rome in 275 B.C at Malventum, which is in that part of southern Italy then known as Apulia. During the battle, the Romans scattered Pyrrhus' elephants by sending pigs coated with grease and pitch and set afire toward the elephant lines.[5] The pigs' anguished cries panicked the elephants.

**Mediterranean World.** In another subdivision of Alexander's empire, Ptolemy I of Egypt began building an army of war elephants with 43 Asian elephants captured at the battle of Gaza in 312 B.C. More significantly, Ptolemy's son (Ptolemy II Philadelphus) began to add African elephants to his corps.[23] These elephants belonged to the now-extinct North African elephant species found along the forested coasts of the Red Sea and in the highlands of Eritrea. In wars with the Seleucids over possession of Syria, African and Asian elephants joined combat on opposing sides. In the battle of Raphia (217 B.C.), Ptolemy's African elephants refused to fight the larger Asian war elephants (although he still won the contest).

The taxonomy of the North African elephants captured by both the Ptolemies and the Carthaginians is much debated and may never be resolved. The only undisputed fact is that they were smaller than the Asian elephants they were deployed against. North Africa and the Near East may seem unlikely habitats for elephants today, but the region was considerably moister and more vegetated until about 2,000 B.C.[23] Asia Minor and Mesopotamia were known as "the land of perpetual shade." Elephants formerly ranged across North Africa, but by the time Carthage was equipping its force, the most readily available elephants were restricted to forested areas to the west between the Mediterranean Sea and the Atlas Mountains (in current-day Algeria).[17,18,22] The small African elephants used in Ptolemy's forces were undoubtedly from the Horn of Africa and the Near East.

Laursen and Bekoff[11] assign the extinct Ethiopian elephant of northern Africa to *L. a. pharaohensis*, a subspecies of the "*cyclotis* division" of *L. africana*. Nowak suggests the forest elephant (*L. a. cyclotis*) could have persisted along the coast of the Red Sea until the mid-1800s.[14] Sukumar raises alternate possibilities that the forest elephant (*L. cyclotis*) expanded into North Africa northward through the Atlantic coastal forests of Africa's west coast or that the small North African elephants were an extinct subspecies of the savannah elephant (*L. africanus*) of eastern and southern Africa.

Further molecular genetic research on subfossil specimens, such as ancient Egyptian ivory artifacts, may someday resolve this mystery.[23]

The best known use of African elephants in war was that of Carthage during the Punic Wars with Rome. Inspired by Pyrrhus' successes, Carthage began building an army of forest African elephants for its campaigns against Rome in 277 B.C.[8] During the first Punic War (264–241 B.C.) a Roman army commanded by Marcus Atilius Regulus invaded North Africa and seized Tunis. The Carthaginian army responded with a force of about 100 war elephants, which were responsible for Roman panic and defeat.

During the Second Punic War (218–201 B.C.), the Carthaginian general Hannibal launched his famous attack on Rome by landing his army of 30,000 men and 37 elephants in Spain and traversing the Alps into the Roman heartland. Most of Hannibal's elephants died by the following year, but he received reinforcements, including elephants, and went on to threaten the gates of Rome. He withdrew to North Africa after a 15-year Italian campaign. Hannibal was finally defeated by Publius Scipio at Zama in 204 B.C. Carthage was forced to abandon the training and use of war elephants in the resulting peace agreement.

While Rome captured its opponents' elephants in many battles, the Roman army never embraced elephants as a war machine. War elephants were used occasionally, but future emperor Julius Caesar doubted their effectiveness in battle and they quickly vanished from the European battle theater following the rise of Imperial Rome.[5,17,23]

**Asia.** During the first millennium A.D., India saw the rise and fall of a variety of Hindu kingdoms. Elephants remained an important part of their military conflicts. War elephants were also employed by the Ghaznavid kingdom to the northwest (in present-day Afghanistan). Elephant inventories of 1,300 in 1024 A.D. and 1,670 in 1031 A.D. are reported.[23] Most of northern India was conquered by Turkish forces in 1192 A.D., leading to the 200-year Delhi Sultanate. By the mid-14th century, the sultanate boasted 3,000 war elephants.[23] In 1398 A.D. an invasion by Amir Timur (= Tamerlane) routed the few remaining war elephants of the Delhi Sultanate. Lacking elephants, Timur had bales of hay strapped to cattle and buffaloes, which were ignited as they approached the elephant line.[5] The retreating elephants devastated their own army.

The Delhi Sultanate was followed by the Mogul Empire. Its greatest emperor, Akbar (1556–1605), built a stable of about 5,000 war elephants, which is reported to have grown to 12,000 during the reign of his son Jehangir (1605–1627 A.D.).[23] Akbar successfully employed his war elephants in many victories, including that against the Hindu stronghold of Chitor in 1567.[5] The astounding numbers of elephants taken from the wild was unsustainable and led to ever-shrinking numbers of wild elephants on the Subcontinent.

The arrival of field artillery on the battlefields of Asia in the 18th century spelled the end of the elephant as an instrument of war. Known as the "tank of the ancient world," the elephant was relegated to supply and support functions. During the Burma Campaign of World War II elephants were used by both sides for transport in mountainous jungle terrain. The Japanese capture of Mandalay was facilitated by a rapid flanking maneuver carried out with elephant transport.[5] Most recently, during the Vietnam War, the Vietcong used elephants to move goods along the Ho Chi Minh Trail.[23,27] So seriously was this viewed by U.S. forces that American fighter-bombers were deployed against these last military elephants.

## Ceremonial Elephants

Because their size makes them impressive, elephants have been used in displays of wealth and power since ancient times. Considerable resources must be devoted to keeping even a small number of captive elephants; hence their public display sent a message about the status of the owner. Elephants were taken as battle prizes by King Shalmaneser III of Assyria (855–824 B.C.).[5] Elephants from Ceylon (Taprobane in ancient times, now Sri Lanka) were being exported to India for war and ceremonial purposes aboard special boats from about 200 B.C.[9] In reciprocal gestures, Sri Lanka's monarchs received gifts of elephants from mainland populations of both India and Burma.

In the Upper Nile Valley of Africa, the kings of Meroe were depicted riding elephants about 400 B.C.[23] The Romans used captured African and Asian elephants primarily in the triumphal processions of returning armies. A darker and perhaps more symbolic Roman use of captured elephants was in their infamous arenas. Sukumar[23] and other sources suggest that public torment and killing of elephants represented the Empire's strength over vanquished foes. Because elephants originated outside the Empire, they were surrogates for enemy peoples. Elephants fought elephants, rhinoceroses, lions, and gladiators for the amusement of the crowds. Gröning and Saller relate a particularly gruesome spectacle sponsored by Pompey in 55 B.C., in which African prisoners of war fought 20 elephants with spears.[5] The usually bloodthirsty crowd was so moved by the elephants' death throes that its sentiment turned against Pompey.

Elephants had long been demanded as tribute from vassal states in India and the Middle East. They soon became spectacular gifts to European kings, whose courts were, sadly, inexperienced in elephant care.[5] The Caliph of Baghdad arranged to transport an Asian elephant to Charlemagne at his court in Aachen in 802 A.D. The animal drowned 2 years later while attempting to cross the Rhine. The next elephant to show up in

Europe was a gift from the Sultan of Cairo to King Fredrick of Sicily (1212–1250 A.D.). This animal fared better and accompanied Fredrick's triumphal march into Milan in 1237. Following the Sixth Crusade, Louis IX of France returned with an elephant, which he presented to his brother-in-law, Henry III of England, in 1255 A.D. In a dubious gesture of hospitality, it was quartered in the Tower of London, where it succumbed within 3 years. Gifts of elephants and other exotic creatures from newly conquered colonies became the fashion among European rulers of the 16th and 17th centuries. Setting the trend, Manuel I of Portugal presented Pope Leo X an elephant named Hanno, who died 2 years later.

## Working Elephants

**Asia.** Although the ancient use of Asian elephants in war and pageantry is noteworthy, an even older and more ubiquitous use of elephants is as a beast of burden. See Figure 2.2. Wylie suggests that the use of elephants for building, logging, and hauling dates to at least 2000 B.C.[27] Because of their size, strength, and intelligence, they have been employed as living machines for many types of heavy duty work. Due to the mundane nature of their tasks, the daily labors of working elephants are little mentioned in historical records. Gröning and Saller cite their work on civic construction projects in Sri Lanka dating from the 5th century B.C.[5] Sukumar observes that the forest tribes of India were experienced in elephant capture and training and were the most likely source for working elephants used by Aryan invaders of the first millennium B.C.[23]

The widespread use of elephants in moist tropical forestry in Asia dates to the middle of the 19th century.[23] The British colonial administration developed extensive bureaucracies to expand the economy and culture of timber harvest by elephants. Not only can elephants move through difficult terrain better than modern equipment, but they cause minimal damage to the forest undergrowth. See Figures 2.3 and 2.4. Teak was an especially prized wood and was harvested in British-controlled Burma (Myanmar) in the late 19th century using thousands of timber elephants.[5] Myanmar is unique in its continued use of about 5,000 timber elephants in its logging industry. See Section 7, "Myanmar," in Chapter 35, "Veterinary Problems of Geographical Concern."

Lair provides an overview of the status of wild and captive elephants in India and 10 other Southeast Asian

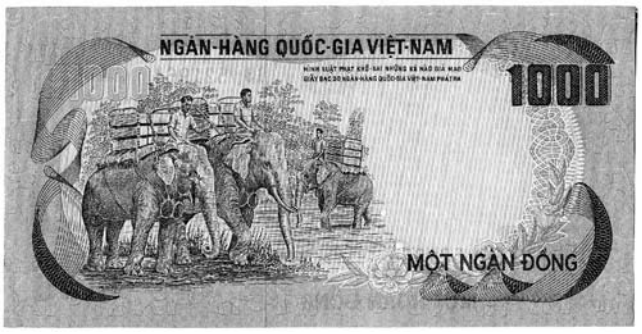


***Figure 2.2.*** *Elephants depicted as beasts of burden on the currency of Vietnam. (Image courtesy of Hank Hammatt.)*

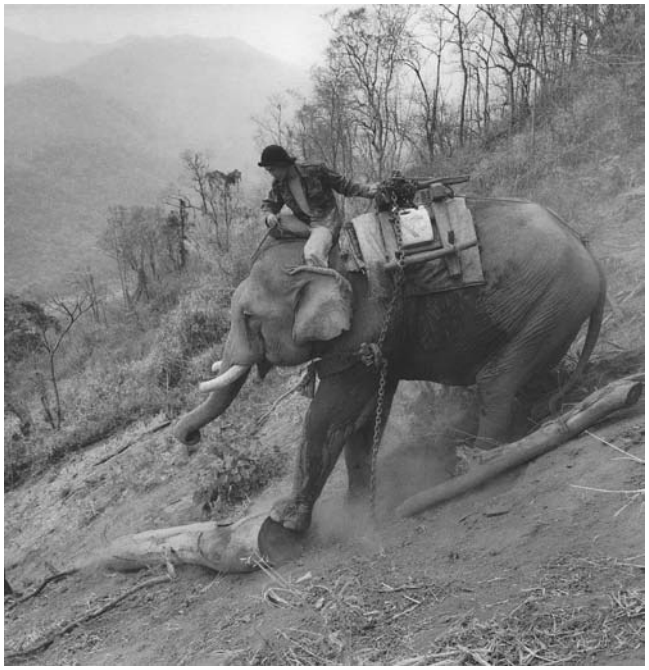

***Figure 2.3.*** *The ability of elephants to work in steep terrain (and the potential for injury) is evident in this photo taken during the dry season in Hongsa District, Sayabouly Province, Laos (2004). (Image courtesy of Philippe Coste.)*

***Figure 2.4.*** *Elephants are still used for logging in Laos and Myanmar, as shown in this photo taken in Luang Prabang district, Luang Prabang Province, Laos (1999). (Image courtesy of Philippe Coste.)*

nations.[10] Countries of particular interest are Indonesia and Thailand.

Indonesia lacked a modern history of captive elephant management. Increasing human populations and clearing of low-elevation forests for commercial plantations on Sumatra resulted in increasing human-elephant conflict in the 1980s. A proposed solution to this problem was the development of Elephant Training Centers (ETCs). The stated intent of these centers was to remove elephants from areas of human-elephant conflict and train them for productive activities. However, neither the intended uses nor anticipated revenues have been realized and the centers have become too costly.[7] Many elephants do not survive the capture process and those that do often sustain unnecessary injuries. Urgent reassessment of the use of ETCs as a solution of human-elephant conflict has been suggested.[6]

Thailand had a long history of captive elephant use in culture and industry. There could have been as many as 100,000 captive elephants in the kingdom at the end of the 19th century compared to about 2,500 in 1998.[5,10] Laws were enacted to protect Thailand's remaining forests in 1989, resulting in loss of employment for 70% of the country's timber elephants.[26] Although increased tourism focusing on elephants may provide some support for displaced elephants and their native handlers, relocation to reserves and training centers in natural habitat (Thai Elephant Conservation Centers) has also been suggested.[26] See Figures 2.5 and 2.6.

**Africa.** Nearly 2,000 years after Carthage trained captive forest elephants for war, King Leopold of Belgium initiated the development of a school for working elephants in the Congo. Initial missteps in 1899 were followed by the successful capture and training of young elephants starting in 1902 under the direction of Commandant Jules Laplume.[19,22] By 1910 there were 35 elephants at the Api elephant station (also called the Elephant Domestication Centre), although it was nearly abandoned during the First World War. In 1919 native handlers (mahouts) from Sri Lanka were brought in to refine training techniques.[22] The station moved to a new location, Gangala-na-Bodia, within Garamba National Park by 1930.[8] At its peak in the 1950s 84 elephants worked at the station doing various jobs, including plowing fields, hauling timber, and pulling carts. Elephant capture and training was abandoned during and following the civil wars accompanying independence in 1960. By 1980 only four trained elephants survived. In 1987 additional elephants were trained for various tasks at the station, including providing elephant rides for tourists.[19]

Randal Jay Moore took up the challenge of returning captive African savannah elephants (*L. a. africanus*) to the Okavango Delta of Botswana in the late 1980s.[5,8] The herd's nucleus of three animals was soon supplemented by an adult elephant and seven calves from Kruger National Park, South Africa.[8] The elephants are driven by African mahouts and offer safarilike rides to

***Figure 2.5.*** *Logging demonstration for tourists at The Elephant Training Center, Chiang-Dao, Chiang Mai, Thailand (2001). (Image courtesy of Hank Hammatt.)*

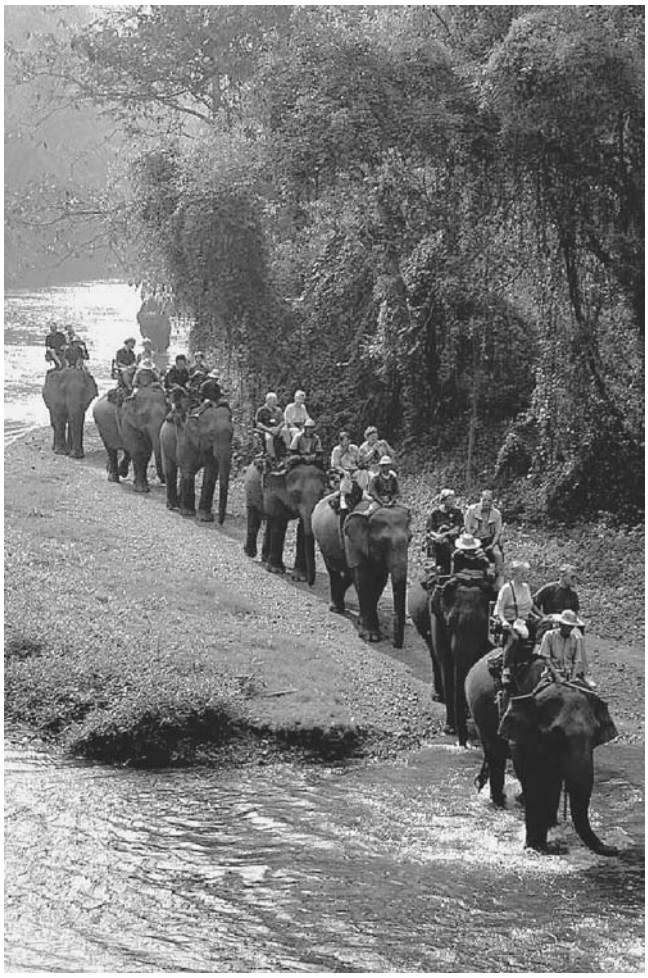

***Figure 2.6.*** *Elephant rides for tourists at The Elephant Training Center, Chiang-Dao, Chiang Mai, Thailand (2001). (Image courtesy of Hank Hammatt.)*

visiting tourists. This experiment is remarkable not only in being the first recorded use of unconfined captive savannah elephants but also by showing considerable promise of developing into a profitable tourist attraction. Each visitor to the Botswana enterprise pays U.S. $3,625 for a 6-day camping safari experience.[8]

### Circus Elephants

The first performing elephants have been traced to courts of Vedic Indian princes before 1000 B.C.[1] In addition to the torture and killing of elephants in Roman amphitheaters mentioned above, Romans also trained elephants to perform tricks startlingly reminiscent of modern circus acts for the amusement of their audiences. Their circus elephants received the best of care and were likely trained by experts from Alexandria. Their repertoire included walking tightropes, carrying dancers on their backs, wearing costumes while mimicking guests at a banquet, and throwing daggers with their trunks.[1,5] The modern circus had its origins in Europe around the turn of the 19th century.[5] At that time human performers and trained animals were integrated into an entertainment program in circuses in England and France. Ironically, although the first elephant arrived in North America in 1796, the large and elaborate circus was an American invention of the mid-1800s. By 1887, the Barnum, Bailey, and Forepaugh circus was able to muster a show of 160 elephants in New York City's Madison Square Garden.[5] Barnum's circus put on a traveling show on a 1901–1902 European tour, which led to increased professionalism and advances among his European rivals. Elephants remain central players in today's circuses, often working to move equipment and set up tents when not performing. In 1995 Ringling Bros. and Barnum & Bailey established the Center for Elephant Conservation in central Florida, U.S. Since 2001 seven Asian elephant calves have been born at the facility, but it is unclear whether this effort will substantially contribute to the goal of a self-sustaining circus elephant population in North America.

### Zoo Elephants

Captive elephants have been kept in animal collections, originally royal menageries, for at least 3,500 years.[4] Elephants persisted in the mountains north of Mesopotamia into historical times. The Sumerian city of Ur (ca. 2000 B.C.) is reported to have maintained them in its zoological gardens.[5] Elephants were also present in collections in Assyria during the reigns of Tiglath-Pileser I (ca. 1100 B.C.) and Ashurnasirpal II (884–858 B.C.). Thutmose III (1504–1450 B.C.) not only hunted elephants, but returned living specimens to his collection of plants and animals at Thebes.[23] The popularity of royal animal collections waned during the Middle Ages, but interest in the collection and display of exotic animals revived following European exploration and the expansion of trade in the 16th century. The royal menagerie at Versailles held African elephants from 1665 to 1681[5] and that at Schönbrunn in Vienna included elephants in 1752.[4]

By the mid-19th century zoos that would be recognizable as such today had emerged. The elephant remains one of the most popular and sought-after exhibit animals for zoos. Sukumar[23] estimates that most of the 1,000 captive African elephants live in western zoos. A similar number of Asian elephants are found in zoos and circuses worldwide. Although zoos argue that elephants provide an opportunity to educate the public about elephant conservation, others suggest that confining such large, social, and ambulatory species in small spaces raises behavioral, veterinary, and ethical concerns. They believe breeding should be halted and elephants in zoos should be phased out.[3]

## ELEPHANT TRAINING

During the Mauryan empire of 3rd century B.C. India, a manual of statecraft called the *Arthasastra* gave explicit directions for keeping and training Asian elephants. Although few mahouts have studied the Sanskrit texts, these ancient training rituals have been passed down through generations of mahouts.[23] Indian mahouts customarily accompanied their charges to remote lands in ancient times, both to control their elephants and to pass along their knowledge to local handlers.

A variety of methods have been used to capture elephants singly or in groups. Primary methods have been pitfalls, noosing, and driving a herd into a strong log enclosure (*keddah* or *khedda*). Chemical darting, often from the back of a trained elephant, is the modern method. Capture often involves a high risk of injury and mortality for elephants.[12,13]

After capture, individuals are removed for taming and training. One form of traditional taming (or "breaking in") involves tying the elephant to trees or other sturdy anchors with ropes attached to opposing front and rear limbs. The ropes are tightened so that the elephant's weight places unnatural stress on the leg joints, causing pain. Food and water are often withheld and fires built to deprive the captive of sleep. After some hours or days of struggle, the person destined to be the main handler will soothe the animal and eventually offer food and drink. The object is not only to have the elephant resign itself to captivity, but to establish the unquestioned control of the mahout over the animals' every movement. When the elephant is reasonably docile, it is taken to bathe while escorted by two elephants (*kumkies* or *koonkies*) specially trained to participate in the capture and training of wild elephants. After the initial breaking in period, it usually takes another 6 months for the elephant to accept the mahout riding on its neck and to respond to about 30 basic commands. Elephants destined for specialized tasks (such as jungle forestry) undergo further training for another 2–3 years.

The traditional training process is described in some detail by Tennent,[25] Jayewardene,[9] and Gröning and Saller.[5]

Elephants in modern zoos are also trained to respond to vocal and visual commands from their keepers; however, most training is accomplished through positive reinforcement. Carl Hagenbeck, a circus entrepreneur from Hamburg, Germany, is largely credited with introducing a gentler training regimen ("tame training") to zoos and circuses in the early 20th century.[5] Because the environment of zoo elephants is more controlled than that of unconfined working elephants, training is usually focused on behaviors that assist with husbandry or veterinary care, as in presenting a foot for pad trimming or toenail filing.

## CAPTIVE ELEPHANTS AND THE FUTURE

Captive elephants have been extensively studied, and often the results have relevance to the conservation of the species.[15,20] Disease diagnosis and control, pheromone analysis, contraceptive methods, and the use of infrasound have all derived or benefited from studies of captive elephants.

The future for captive elephants is not promising. Elephants in zoos and circuses are not breeding at a rate necessary to sustain these populations. Breeding success is higher in working elephants of some Southeast Asian countries, [23,24] but still not sufficient to maintain the size of the largest current captive population in Myanmar. Efforts to reverse the decline in captive population numbers may be restricted by the social needs of elephants. Taylor and Poole[24] suggest that the key is larger herd groups and continued access between males and females. Given the space requirements of elephants and the small average numbers of elephants per zoo, this may not be achievable.

The large numbers of Asian war and working elephants of the past were captured from shrinking wild populations. Human population growth in the past century has led to unprecedented loss and fragmentation of habitat for wild elephants. Conflicts between growing human populations and elephants displaced from their former habitats are nearly always resolved in favor of humans. It may not be ethical to maintain captive populations of Asian elephants by capture from the wild. Although the status of the African elephant is not as critical, increasing human population growth and appropriation of elephant habitat worldwide portends fragmented and declining populations of all three species. Soulé concluded that "the evolution of large, terrestrial organisms in the fragmenting tropics is all but over."[21] Elephants have been human servants for half of the Holocene. If elephants are to survive the present human-driven extinction spasm, sufficient habitat for viable wild populations must be maintained in Africa and Asia.

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# 3 Laws and Legislation

Denise M. Sofranko

## INTRODUCTION

Few of the worldwide variety of laws and legislation pertaining to animals specifically address elephants. Most are general, relating to conservation and trade in wildlife or animal welfare of captive animals. This chapter highlights significant legislation or laws that affect both wild and captive elephants. This is by no means a complete list or discussion of all laws pertaining to elephants. This information can be treated as a starting point from which interested individuals may delve deeper into individual countries or regions as needed.

Despite laws to protect wild and captive elephants, there are barriers to enforcement in many areas. Underfunding, lack of education or information, corruption, and the continuing thirst for ivory and other products fosters illegal trade. Lack of resources, inadequate coverage, and confusion about the law might hamper legal protection for captive elephants. The descriptions and citations of law are made in this chapter with minimal comment on their enforceability. Although every country could not be included, the range states where elephants still exist in the wild are listed plus other countries where there is a significant elephant population in captivity. Pertinent laws for selected countries are listed and, where possible, a short summary of the law is included.

## CONVENTION ON INTERNATIONAL TRADE IN ENDANGERED SPECIES (CITES)

The Convention on International Trade in Endangered Species of Wild Fauna and Flora (CITES) is a treaty among member nations that addresses trade (import and export) of certain animals, including elephants.[20] Laws that specify actual handling or welfare of elephants are found at national and state or provincial levels (Table 3.1).

Eighty countries agreed to the Convention in March 1973 in Washington D.C. There are currently over 160 members. CITES followed a resolution of the IUCN (International Union for the Conservation of Nature, currently known as the World Conservation Union) in response to a need to regulate wildlife trade, including Asian and African elephants. CITES is a cooperative agreement among countries (*Parties*) that agree to use their own legislation to ensure that the treaty is implemented. The CITES Secretariat is located in Geneva, Switzerland and is the coordinating body of the Convention. The Secretariat is administered by the United Nations Environment Program. A Conference of Parties meets every 2–3 years to renew implementation of the Convention, make changes in the appendixes, review progress in conservation, and adopt a budget.

Species of animals under CITES are listed in three appendices. The Asian elephant (*Elephas maximus*) is included in Appendix I. The African elephant (*Loxodonta Africana*) is contained in Appendix I except for those elephants in Botswana, Namibia, South Africa, and Zimbabwe, which are listed in Appendix II. These lists may be changed either by the Parties at their regular Conferences or between Conferences by mail, following prescribed rules.

### Appendix I

Appendix I "shall include all species threatened with extinction which are or may be affected by trade. Trade in specimens of these species must be subject to particularly strict regulation in order not to endanger further their survival and must only be authorized in exceptional circumstances."

### Appendix II

Appendix II includes "(a) all species which although not necessarily now threatened with extinction may become so unless trade in specimens of such species is subject to strict regulation in order to avoid utilization incompatible with their survival."

In 1989, a compromise known as the Somali Amendment was proposed. This proposed that all elephant

**Table 3.1.** Legislation Pertaining to Captive Elephants in Selected Countries

| Country | CITES | Legislation | Comments | Reference/Further Info |
|---|---|---|---|---|
| Austria | 1982 | | A 2004 law bans the use of wild animals in circuses. | 13 |
| Germany | 1976 | Animal Welfare Act 1998 | Section II (Article 3) prohibits certain training and handling practices, including the use of electricity to restrict or redirect behavior; training which may cause pain, suffering, or harm; and performances beyond an animal's strength or capacity. | 43 |
| Netherlands | 1984 | The Animal Health and Welfare Act 1992 | This act prohibits "inflicting unnecessary pain or injury, or damaging an animal's health or welfare, or the withholding of essential care" (applies to wild and domestic animals). | 25 |
| Sweden | 1975 | Criminal Law 1857; The Animal Welfare Act 2002; The Animal Welfare Ordinance 2002 | The 1857 law prohibited abuse; the Welfare Act and Welfare Ordinance have sections specific to zoos and circuses requiring operating permits. | 54 |
| Switzerland | 1975 | The Swiss Federal Act on Animal Protection 1978; The Swiss Animal Protection Ordinance 1981 | The Ordinance includes rules for the professional keeping of wild animals. | 43 |
| Norway | 1976 | Animal Welfare Act (revised 2003) | Except with permission (such as for zoos and some circuses), it is illegal to exhibit animals in public. | 43 |
| Brazil | 1975 | The Federal Environmental Crimes Law | This law prohibits abuse; includes confinement that would be considered cruel, overwork, wounding or injuring an animal, and using physical punishment to train; includes some local laws that restrict the ownership of exotics. | 43 |
| New Zealand | 1989 | Animal Welfare Act 1999 | The Act has three parts: Core Obligations of People, Codes of Welfare, and Regulations; specific codes were written in 2005 for the care of animals in zoos and circuses. | 15 |

populations be placed in Appendix I of the **CITES** Convention, but that a mechanism should be created whereby any Range State with a healthy elephant population could apply to CITES to have its elephants placed in Appendix II subject to a series of verifications and a vote of the Parties to CITES.

The 7th Conference of the Parties to CITES approved a worldwide ivory trade ban in September 1989. In 1997 some of the populations (Botswana, Zimbabwe, and Namibia) of African elephants were transferred back to Appendix II, with specific conditions assigned. These countries were allowed a one-time conditional sale of ivory. The conditions were met and a sale took place in April of 1999.

In 2000, the South African elephant population was down-listed to Appendix II with no ivory sales approved, and proposals from Botswana, Namibia, and Zimbabwe to continue to sell ivory were withdrawn. In 2002, Botswana, Namibia, South Africa, and Zimbabwe proposed to sell off stocks of raw ivory and asked for the establishment of future quotas. This was accepted with certain conditions and with all proceeds to go to programs benefiting African elephants.

In 2004, it was decided that conditions for the one off-sale of ivory from Botswana, Namibia, South Africa, and Zimbabwe had not been met and there would be no sale until further notice (Thirteenth meeting of the Conference of the Parties Bangkok, Thailand, 2–14 October 2004).

## The Elephant Trade Information System (ETIS)

The Elephant Trade Information System (ETIS) is a comprehensive system to track illegal trade in ivory and other elephant products. It was proposed at the 10th meeting of the Conference of the Parties (1997) and amended at the 12th. The objectives include assessing whether the changes in the listing of the elephant populations and legal trade in ivory are related to trends in illegal trade. This system began with a database of illegal ivory seizures and has progressed to a more sophisticated system. The database is meant to aid management and enforcement.

## Monitoring the Illegal Killing of Elephants (MIKE)

The poaching of elephants prompted CITES to pass a resolution in 1997 to monitor African and Asian elephants over their entire range. Monitoring of Illegal Killings of Elephants (MIKE) was launched 2 years later in Africa.

MIKE is similar to ETIS except that it is meant to monitor the illegal killing of elephants and illegal ivory trade. Objectives also include assessing whether trends in illegal killing of elephants are related to the changes

in listings in the appendixes and to legal ivory trade. Like ETIS, a database is used to aid management and enforcement. As of this writing MIKE is active in 29 countries in Africa and in 5 of the 13 Asian range states (India, Sri Lanka, Nepal, Bangladesh, and Bhutan).

## COUNTRY REGULATIONS

### India

India became a signatory to CITES in 1976. There are approximately 3400–3600 captive elephants in 24 states in India.[49] Approximately 75% are owned by private individuals and 14% by the Forest Department, with the rest held by temples (6%) and circuses and zoos (5%).[16] Laws in India pertaining to elephants go back as far as the 3rd century B.C. The Indian Forest Act (1878) gave provinces the power to regulate the killing or capture of elephants and other wild animals and was followed by the Elephant Preservation Act of 1879.[49]

The most important modern law governing elephants is the Wildlife Protection Act (WPA) of 1972, amended in 2002. In the original version, the elephant was in the same category as cattle. A 1991 amendment replaced cattle with livestock but excluded elephants from that category. This had the unintentional effect[16] that elephants, unlike livestock, were no longer required to receive prophylactic vaccinations. A vehicle, as defined in the Act, could also include elephants. With the inception of CITES and India's becoming a signatory to that Convention, the WPA was changed to reflect the provisions of that body. Prior to that, the elephant in India was considered a "Special Game" animal and could be killed, captured, or traded with the proper license. Ivory was not regulated under the 1972 Act.[16]

Under the influence of CITES, the elephant was placed under Schedule I in 1977, and export of elephants and ivory from India was banned in 1978. Domestic trade in ivory was banned in 1986.[16] There is a provision in the WPA for second-generation captive elephants to be exported under CITES permits. Zoos are also given special status and are allowed to import elephants under specific conditions.

Currently, hunting and capturing of elephants is banned under the WPA except under circumstances where the elephant is considered dangerous or is in a state of suffering. Any killing of a dangerous elephant is done only if the Chief Wild Life Warden (CWLW) determines that the elephant can't be captured, tranquilized, or relocated. A captured elephant cannot be kept in captivity unless the CWLW determines that it can't be rehabilitated and released. The CWLW has the authority to determine whether the captive conditions are adequate.[30]

There is a provision for hunting or capturing elephants for scientific study or education or to collect for zoos (WPA, 1992). The current Act provides for Sanctuaries and National Parks to protect wild flora and fauna and regulations on activities within those protected areas. Private elephant owners must have permits from the CWLW, and permission must be granted to move or transport an elephant.[16]

The Indian Forest Act of 1927 has provisions to protect elephants in protected forests or reserves. Elephants are recognized and protected as "forest produce." Permits are required to move the elephants under this Act.[49]

Under the Recognition of Zoo Rules, 1992, a zoo must apply to the Central Zoo Authority (CZA) to be recognized. The rules under this law are based on the 1972 WPA. The CZA is appointed by the Central Government and is responsible for specifying minimum standards for housing and veterinary care, identifying endangered species for captive breeding, coordinating the training of zoo personnel, coordinating captive breeding research, and educational programs.[30]

India's Prevention of Cruelty to Animals Act (1960) applies to domestic and nondomestic captive animals, including elephants. An Animal Welfare Board appointed by the Central Government administers the law. Although cruelty is not specifically defined, Chapter III of the Act lists actions considered cruel. Chapter V addresses performing animals and contains rules for registering as an exhibitor or trainer.[35]

Rules written under this Act include those for Performing Animals (1973 and 2001) and Capture of Animals Rules (1979). The Performing Animals Rules specify conditions for registration, travel restrictions, feeding and watering, work periods, work conditions, etc. (Prevention of Cruelty to Animals Act, 1960): "The Indian elephant now enjoys much more legal protection than ever before. But the enforcement of the laws leaves much to be desired."[16]

### Bangladesh

Bangladesh became a signatory to CITES in 1982. There are an estimated 200 wild and 93 domestic elephants in Bangladesh.[36] The Bangladesh Wild Life Preservation Order (President's order No. 23, 1973, amended in 1974) protects wildlife (including elephants) but does not protect captive elephants. A Wild Life Advisory Board is set up under the Order. There are three schedules; elephants are in Schedule 3, protected animals. The Order gives the government the power to designate national parks or game reserves.[30] The Forest Department is in charge of registering domestic elephants, but this is not well enforced.[36]

### Sri Lanka

Sri Lanka became a signatory to CITES in 1989. There are an estimated 3160–4405 wild elephants[39] and 400–600 in captivity[53] in Sri Lanka. In 1872, in response to a decline in the game population in Sri Lanka, the British government passed a law (the Ordinance) to prohibit the shooting of buffalo and game from April to September. In 1891 another law (the Ordinance to prevent wanton destruction of elephants, buffalo, and other game) was

passed that specifically mentioned the elephant. Although it didn't prohibit elephant hunting, it did limit the times and methods with which it could be done.[38]

In 1937, the first version of the Sri Lanka Fauna & Flora Protection Ordinance was passed. This law remains in place today as amended in 1993. The law makes provisions for the establishment of National Reserves and Sanctuaries and laws governing their use. The amended version also makes provisions for the establishment of elephant orphanages. The law is administered by the Department of Wildlife Conservation and Forest Conservation Department. Part II pertains to wild and "tame" elephants and buffalo. The 1970 version included provisions to issue licenses to capture, drive off, or even kill elephants if there was serious danger to life or property, but these were rescinded in 1993.

Section 22 of the 1993 amended Ordinance prohibits the export of tusks/tushes or any other elephant part or product, but there is no prohibition against the export of live elephants. There is an export duty and a permit is required.[30]

Sri Lanka's Prevention of Cruelty to Animals Ordinance of 1907 was seen as inadequate by many and a new draft Animal Welfare Act has been prepared and is undergoing public comment and finalization (personal communication, Jayantha Jayewardene, Sri Lanka, March 2005).

The National Zoological Gardens Act provides for the administration and management of the National Zoological Gardens. A Zoo Development and Welfare Fund is established under the Act. National Zoological Gardens include the public aquarium, zoo farm, and elephant orphanage.[30]

### West (Peninsular) Malaysia

Malaysia signed on to CITES in 1978. In 2000, the population of wild elephants in Malaysia was estimated to be 1200–1500. There are about 36 domestic elephants.[22] The Department of Wildlife and National Parks, Peninsular Malaysia (DWNP) is a federal department under the Ministry of Science Technology and Environment. The Protection of Wildlife Act 1972 (Act 76), amended in 1988, includes elephants. This Act applies only to West Malaysia. The Director General of Wild Life and National Parks is appointed to administer the law. The Act gives the ruler or governor of a state the power to declare state land as a wildlife reserve or sanctuary or define or alter the boundaries. Permits must be obtained to enter wildlife sanctuaries or reserves.

Animals are categorized in different schedules; elephants are in Schedule Two: Protected Wild Animals. Special permits may be granted to shoot, kill, or take a protected wild animal if it is in the interest of scientific research, the animal is damaging crops or property, or the animal is a danger to human life. Outside these parameters, a person convicted of killing a protected species may be fined or imprisoned or both.[30]

Domestication of elephants in zoos and safaris and by individuals is allowed with a permit from the DWNP. Only the DWNP may capture elephants. Cruelty is covered by the Act and is punished by fines or imprisonment or both. The DWNP prescribes guidelines for zoos, safaris, and private elephant owners.[22]

### Malaysia, State of Sabah (Northern Borneo)

Elephants are listed in Schedule 2, Protected Species of Animals and Plants—Limited Hunting and Collection Under License, in the Wildlife Conservation Enactment, 1997, of Sabah. A Department of Wildlife oversees protection of animals and hunting, establishment of sanctuaries and conservation areas, zoo permits, possession and trade, utilization of wildlife, enforcement, and penalties. In the case of zoos, the Director has the authority to oversee building design and construction, provision of veterinary care, sanitary conditions of the animals, and prevention of escape plans. Violators of the Act may be fined and/or disqualified from holding a permit for a period of time, or they may be imprisoned.

Persons who operate wildlife tour companies or breed, rear, or keep animals are required to have a permit. The Director is responsible for issuing licenses for hunting protected animals and for establishing limits on the number of each species that can be hunted.[30]

### Myanmar

Myanmar became a signatory to CITES in 1997. Currently, there are an estimated 3000 state-owned elephants, 2000 privately owned elephants, and 5000 wild elephants.[62]

The Elephant Preservation Act (1879) regulated hunting and capture. The Burma Wildlife Protection Act (1936, revised in 1956) banned hunting without a license.[10] The Protection of Wildlife and Wild Plants and the Conservation of Natural Areas Law, 1994, is the predominant law protecting elephants in Myanmar. Elephants have been listed as a completely protected wildlife species. Violations may result in confiscation of animals or parts of animals, fines, or imprisonment.[9]

Under the Essential Supplies and Services Act (Burma Act XLVII, 1974) and the Elephant Registration Act (1951) private and Myanmar Timber Enterprise (MTE) elephants must be registered with the Forest Department. MTE elephants are state owned and used in the timber industry. MTE elephants are registered with the MTE and branded to further identify them.[10]

### Cambodia

Cambodia became a signatory to CITES in 1997. From a 2000/2001 survey, it is estimated that there are 300–600 wild elephants and 162 domestic elephants. Domestic elephants belong to Phnong clans made up of a number of

families, and those elephants may be used by any of the families and are passed on to the next generation. There is no national registration, but some provincial level registration is ongoing. The Department of Forestry and Wildlife is planning a national registration program.[23]

Under the Law on Forestry 2002, there is a provision for wildlife to be under the management, research, and conservation of the Forestry Administration. There are three wildlife categories: Endangered species, Rare species, and Common species. The Ministry of Agriculture, Forestry and Fisheries determines the criteria for each category, issues permits, and makes rules on related activities. There is no listing of the species in the Law. The Law on Environmental Protection and Natural Resource Management (1996) mentions wildlife and outlines principles with no specifics. The 1993 document, Regulations on the Creation and Designation of Protected Areas, describes specific areas (with a listing and a map) to be designated as national parks, wildlife sanctuaries, and protected landscapes.[30] There are no specific laws covering domestic elephants, and the wildlife laws are confusing and poorly enforced.[23]

## Nepal

Nepal became a signatory to CITES in 1975. In 2000, there were an estimated 92–113 wild elephants,[40] but current estimates are 57–95.[37] There were about 174 captive elephants in 2003, of which 50% are government owned.[62] The National Parks and Wildlife Conservation (NPWC) Act 2029 (1973) and its 4th amendment 2049 (1993) include wild elephants as a protected species in Appendix I. Capture of wild elephants is prohibited. Killing or wounding a wild elephant or buying a trophy is a violation and may be punished by imprisonment and/or fines. There is a reward system in place for anyone providing information on violators. The Royal Nepalese Army is empowered to enforce this Act.

Although there were laws under the Elephant Management Rules 2022 (1966) to protect domestic elephants, this law has been repealed and there is nothing in the NPWC Act to protect domestic elephants.

Under the Civil Service Act 2049 (1993) there must be three people caring for each elephant in the government elephant camps. There are specifics in the Act for food and water as well. There is a registration system but it addresses only government-owned elephants and their naming. Baby elephants born into captivity are assigned a keeper by the government.[40] Elephants are not allowed to be in National Parks except for jungle safaris, according to a policy of September 2003. This prohibits the private owners from grazing their elephants inside the park.[62]

## Vietnam

Vietnam became a signatory to CITES in 1994. In July 2004, survey results estimated that the domestic herd had been reduced to 62 and the wild herd numbered only 40–50 elephants.[31] The most common reason for the continuing drop in the population of elephants in Vietnam is hunting and poaching for the illegal wildlife trade. There are still problems with elephants being illegally sold across the border to Laos PDR and Cambodia.

Government Instruction 143/TTG (1960) on the prohibition of elephant hunting and Decree No. 39/CP (1963) on temporal regulations of Vietnam for hunting forest birds and animals both pertain to the protection of wild elephants. Decree No. 18/HDBT (1992) contains the schedule for rare fauna and flora and provisions for their management and protection. Elephants are listed in Group IB. Hunting, killing, and selling are strictly prohibited.[21]

The Law of Forest Protection and Development approved by the National Assembly of the Socialist Republic of Vietnam in 1991, followed by the Instruction of the Ministry of Forestry regarding promotion of wildlife protection in 1994, has provisions for spreading awareness and disseminating information about the law, prohibiting commercial establishments from serving wildlife as food, prohibiting the selling of products of protected wildlife, and penalties for violators of the law. It has instructions to observe and follow the provisions of CITES and the Government of Vietnam when exporting or importing wild vegetation or animals and their products.[30]

## Sumatra, Indonesia

Indonesia became a signatory to CITES in 1979. The Sumatran elephant (*Elephas maximus sumatranus*) is a subspecies of the Asian elephant found only on the Island of Sumatra, and like many of the other populations in this region of the world, the numbers are declining. Numbers of wild elephants in 2000 were estimated to be between 2085 and 2690. The number of "domesticated" elephants was estimated at 362 in 2000.[34] The decline in the elephant population, due largely to hunting, resulted in a 1931 law to protect them.[37]

This was renewed in 1972 by the Minister of Agriculture and then amended in 1999 and applies to both wild and domestic elephants. It prohibits hunting, trading, and keeping elephants or parts of elephants without a government permit. It provides for fines and imprisonment for violators.[34]

In response to human-elephant conflict, the government established Elephant Training Centers in 1986. These were meant to train problem elephants for logging, patrol work, and tourism. In 1995, a regulation was made that a fee be required of a person or an institution that used or kept elephants from the Training Centers.[34]

## Laos PDR

Laos PDR became a signatory to CITES in 2004. Population estimates are variable. Wild elephants number 200–500 in one report[41] and <1000 in another.[55] The Livestock and Fisheries Division estimates that there

were about 864 domestic elephants in 2000.[47] The government has established the National Protected Area (NPA) system that covers 21% of the country. The management of these areas is the responsibility of the Ministry of Agriculture and Forestry (MAF) with long-term biodiversity conservation being the primary objective. There are a number of laws that have provisions that apply to wild elephants. The Decree of the Council of Ministers No. 185/CCM, in Relation to the Prohibition of Wildlife Trade, 21 October 1986, prohibits the export of wildlife, and the Decree of the Council of Ministers No. 118/CCM, on the Management and Protection of Aquatic Animals, Wildlife and on Hunting and Fishing, 5 October 1989, defines wildlife as state property, allows import/export of wildlife with specified authorization, and prohibits hunting. The Decree of the Prime Minister No. 164, 29 October 1993, has provisions for the establishment of protected areas, and Order 54/MAF on the Customary Rights and the Use of Forest Resources, 7 March 1996, and the recommendation 377/MAF on the Customary Use of Forest Resources gives local people the right to use forest resources for subsistence. Decree 1074 of the Ministry of Agriculture and Forestry, 11 September 1996, prohibits trade in wildlife and hunting of protected species.[47]

## Thailand

Thailand became a signatory to CITES 1983. There are an estimated 1000–1500 elephants in the wild in Thailand. A recent survey of the domestic elephants numbers them at approximately 2343 in 73 elephant camps and villages.[57] In 1921, under the Wild Elephant Act, wild elephants became the property of the government. The Act forbids hunting for sport. The Wild Animal Preservation and Protection Act of 1960 provided for wildlife sanctuaries and nonhunting areas.[41]

The Wildlife Reservation and Protection Act, also referred to as the Wildlife Protection Act of 1992 and the Act for the Conservation and Protection of Wildlife B.E. 2535, 1992, establishes a Committee on Wildlife Conservation and Protection (chaired by the Minister of Agriculture) that has the power to designate wildlife conservation areas and list protected species. Hunting or attempted killing of protected wild animals is not permitted unless for education or research. These activities may be permitted by the Minister of Agriculture. Wild animals cannot be kept except for public zoos (permission must be granted by the Director General of Forestry or Fishery). This Act does not cover domestic elephants.[30] The Draft Animal Act of 1939 also includes a provision for registration. There is no provision for treatment of the elephants.[41]

## China

China became a signatory to CITES in 1981. There are estimated to be about 250 elephants in the Yunnan province of China.[55] The Law of the People's Republic of China on the Protection of Wildlife (1988) delineates the general principles of wildlife protection and administration of wildlife in the PRC. Wildlife is considered the property of the government. The law is implemented by the Wildlife Protection Law implementing Regulations of September (1993) and Wild Land Life Protection Regulations (1992). The Forestry Department administers the Act and conducts surveys of wild land animals. Wild land animals refers to animals that are "precious" or "being endangered" with high economic or scientific value. Hunting or killing of these animals is prohibited except with special permits. A domestication and breeding license is required for domestic species. Sale, purchase, or use of these animals requires a permit from the government. The law also requires that the government compensate people for losses due to endangered animals.[30]

Although there was a law proposed and later withdrawn in 2004 that would have provided for animal welfare regulations, there are currently no general animal welfare laws to protect animals in captivity.[19]

## Kenya

Kenya became a signatory to CITES in 1979. The current population of elephants in Kenya is estimated at around 30,000 according to the Kenyan Wildlife Service.[46] This is an increase of about 3000 in the last few years.

The Forest Act of 1942 gave the government the power to set aside protected areas, and hunting animals was prohibited in those areas except with a permit from the Chief Conservator in consultation with the Chief Game Warden. The Wildlife (Conservation and Management) Act of 1976 was amended in 2004. The original Act banned poaching and reckless killing of wild animals. The Act makes provisions for National Parks, National Reserves, and Local Sanctuaries; control of hunting; regulation of trophies; and enforcement. The Act further provides for the establishment of a Wildlife Conservation and Management Service (The Service) as a Department of the Government. National Parks shall be managed and maintained by the Director of Wildlife Conservation and Management. The Minister may, after consultation with the competent authority, declare National Reserves, Local Sanctuaries, and Protection Areas. Protection areas are areas adjacent to a National Park, a National Reserve, or a Local Sanctuary and are established for the purposes of protection of fauna and flora in such areas. The regulation of game licenses and other hunting issues is prescribed in the Act.[30]

The new controversial amendment to allow sport hunting and private ranchers to kill wildlife that drift into their land was enacted in December 2004.[4]

## South Africa

South Africa became a party to CITES in 1975; elephants are listed under Appendix II. The estimated number of

wild elephants in South Africa in late 2004 was 17,000, with 12,000 of them in Kruger National Park.[60] The National Parks Act of 1962, repealed by an Act in 1976 and last amended in 1998, sets provisions for declaring National Parks, regulates hunting, and provides for rights of certain people residing in the parks. The Act, which provides for a National Parks Board to oversee the parks, declares the following: "The object of the constitution of a park is the establishment, preservation, and study therein of wild animal, marine, and plant life and objects of geological, archaeological, historical, ethnological, oceanographic, educational and other scientific interest and objects relating to the said life or the first-mentioned objects or to events in or the history of the park, in such a manner that the area which constitutes the park shall, as far as may be and for the benefit and enjoyment of visitors, be retained in its natural state."

The National Parks Act also prescribes penalties for violators consisting of fines and/or imprisonment.[30]

Provincial Nature Conservation Authorities have regulations pertaining to privately owned elephants in reserves.

### Nigeria

Nigeria became a signatory to CITES in 1975. There have been no recent accurate elephant counts. A population of about 478 was estimated in the 1990s.[17] The Endangered Species (Control of International Trade and Traffic) Act specifies animals for which hunting, capture, or trade is prohibited (First Schedule) and others (Second Schedule) where a permit is required. Immature elephants are listed under the First Schedule and mature elephants are listed under the Second.

The Wild Animals Law of 1963 was amended and implemented in 1975. The Law details appointment of a Regional Game Warden and Game Protection Officers, provides for animals to be placed in Schedules reflecting their status as endangered species, and assigns penalties for violations. The Minister for Animal and Forest resources may grant a hunting permit for prohibited animals for scientific or essential administrative reasons.

The captive animal regulations of this law prohibit anyone from keeping any prohibited, protected, or exotic animal in captivity without a permit. A license may be refused if the person cannot make adequate provisions for the health of the animal.[30] The National Park Service Decree of 1999 led to the creation of the National Parks Governing Board and the creation of the Department of National Parks.[7]

### Zambia

Zambia became a signatory to CITES in 1981. A 2003 survey by the African Wildlife Foundation estimated that Zambia has 1423 elephants.[3] The Forests Act of 1999 establishes a Forestry commission and its functions. It repeals the Forests Act of 1973. The International Game Park and Wildlife Act last amended in 1994 gives the President the authority to establish National Parks and implements the National Parks and Wildlife Act of 1991. The National Parks and Wildlife Act, 1991, replaced the National Parks and Wildlife Act of 1971. In 2000, the Zambia National Parks and Wild Life Service was reorganized into the Zambia Wildlife Authority. This transition led to an increase in elephant poaching.[17]

### Zimbabwe

Zimbabwe became a signatory to CITES in 1981, and its elephants are listed in Appendix II. The number of elephants in Zimbabwe is under some debate, with government officials estimating it at more that 100,000; others say it is 60,000 at its highest.[64] The Environmental Management Act of 2002 provides for the sustainable management of natural resources and protection of the environment. The Act amends, among others, the Parks and Wildlife Act of 1996. Unless permission is granted by the Minister of Environment and Tourism, hunting, removing, or selling any animal from the Parks is prohibited. The Minister also has control over Sanctuaries, Recreational Parks, and Safari Parks. Like similar laws, the Act describes legal action to be taken against offenders. The elephant is not listed as a "Protected Animal" but as a "Dangerous Animal" in the Act.[30]

### Ghana

Ghana became a signatory to CITES in 1976. Estimates of the elephant population ranged between 1000 and 2000 in 2001.[17] Others have estimated the population at <1000.[42] In 2000, Ghana produced a strategy for the conservation of elephants.[17] The Wildlife Animals Preservation Act, 1961 (No. 43 of 1961), amended in 1983, allows the Minister to appoint honorary game officers. They may carry out functions for purposes of this Act and have powers equal to game officers. The Act addresses the collection of specimens for scientific purposes and prohibits anyone from exporting any trophy from Ghana unless in possession of a certificate by a Superior Police Officer. Prohibited hunting methods are described and game officers are given the power to arrest without a warrant. Regulations on trophy hunting, exporting, and penalties were written in 1971 (Wildlife Conservation Regulations, 1971, L.I. 685) and amended in 1989. They also provide rules on penalties for violators. Elephants are listed as "Completely Protected."[30]

The Wildlife Division of the Forestry Commission is charged with conserving the wildlife resources for Ghana (Forestry Commission Act, 1999). The original Game Branch of the Forestry Department, now the Department of Wildlife, was established in 1965. The Ghana Wildlife Conservation Policy was adopted in 1974. There are 16 wildlife conservation areas overseen by the Department.

The Forestry Department was first established in 1909. Timber production is the main function of the

280-plus reserves in Ghana, but environmental and ecological stability is also an objective (Ntiamoa-Baidu 1995). The Forestry Commission Act, 1999 (Act No. 571 of 1999), establishes a Forestry Commission that is responsible for protection, development, management, and regulation of forests and wildlife.[30]

### Botswana

Botswana became a signatory to CITES in 1978. The current population is estimated to be over 100,000,[6] with some estimating over 120,000.[17] The Wildlife Conservation and National Parks Act 1992, amended in 1993, implements CITES and other international conventions. The Act designates specific areas as National Parks and gives power to the President to declare any state or bequeathed land as a national park. It also gives the President the power to name game reserves and sanctuaries and private game reserves as well as wildlife management areas and controlled hunting areas. Control, management, and maintenance of national parks is the Minister's responsibility. The Act lists prohibited activities in the National Parks, including removing animals and entry without permission. Rules for hunting are described as well as penalties for violators.[30]

### The United Republic of Tanzania

The United Republic of Tanzania became a signatory to CITES in 1980. In 2000, the population was estimated at 60,000.[1] The Forest Act of 2002 provides for the management of the forests (both public and private) in Tanzania and requires the formulation of official management plans that include the management of wildlife. It includes provisions for granting permits for private use of the land within the forest and for transferring some of the management authority to other local or regional entities, such as villages, under specific conditions.

The Wildlife Conservation Act No. 12 of 1974 establishes protected areas, describes requirements for hunting (must have proper permits) and rules for trophy registration and sale, and sets penalties for violators. The National Parks Ordinance, 1959, last amended in 1975, gives the Tanzania National Parks Authority (TANAPA) jurisdiction over National Parks. A new Wildlife Policy has been adopted to address problems in the management of the parks, use of wildlife resources, sharing rights with local communities, and sharing benefits from wildlife uses and resources.[51]

### Namibia

Namibia became a signatory to CITES in 1991. The elephant population is estimated to be 15,363.[52] The Forest Act of 2001 (No. 12 of 2001) establishes a Forestry Council and consolidates the laws relating to the management of forests and products. The Nature Conservation Ordinance of 1975 (No. 4 of 1975), amended in 1996 with the Nature Conservation Amendment Act, consolidates the laws for the management of wildlife and establishing game parks and nature reserves, as well as the control of problem animals.[30]

### Ethiopia

Ethiopia became a signatory to CITES in 1989. There are estimated to be only about 800 elephants left in Ethiopia.[14] In 1909, Emperor Menilik II issued a notice that hunting could not be done without a permit. A Wildlife Act of 1944 was passed with the main aim to control hunting, but there is still very limited protection of elephants inside national parks and none if they are outside those boundaries. In 1992 hunting was prohibited but has been resumed.[17]

### Cameroon

Cameroon became a signatory to CITES in 1981. There were an estimated 27,600 elephants in 1997.[17] The Forestry and Wildlife Law (1994), under the jurisdiction of the Ministry of Forestry and Wildlife, is strict regarding threatened species. Anyone found with ivory that has not been declared faces severe punishment.[45] The 1994 Act is based on a model of local community involvement. The subsequent Decree of Application on Wildlife (Wildlife Decree) of 1995 has provisions for community hunting zones, shared benefits from wildlife resources, and buffer zones.[26]

### Malawi

Malawi became a signatory to CITES in 1982. There have been no formal surveys of elephants since 1997, but the population is estimated at a little over 2000.[17] The National Parks and Wildlife Act of 1992 implemented by National Parks and Wildlife (Control of Trade in Live Animals) Regulations, 1994 (Government Notice No. 81), requires a permit for hunting or possessing wildlife. A Chief Parks and Wildlife Officer administers the Act, which provides for the establishment of national parks and reserves and wildlife impact assessments. The National Parks and Wildlife (Wildlife Ranching) Regulations, 1994 (Government Notice No. 82), describes the rules for wildlife ranching and has provisions for release into the wild, inspections, harvesting, record keeping, etc.[30]

### Mozambique

Mozambique became a signatory to CITES in 1981.

In 1999, there were an estimated 17,000–19,000 elephants. Commercial ivory hunting was banned in 1960.[17] The Forest and Wildlife Act (No. 10/1999), calls for protection, conservation, and sustainable utilization of forest and wildlife resources. Decree No. 12/2002, approving the implementing of the Regulation of the Forestry and Wild Fauna Act, implements the Act of 1999.[30]

### Uganda

Uganda became a signatory to CITES in 1991. Surveys of the elephant population taken over several years estimate

that there are over 2000 elephants in Uganda.[17] The Uganda Wildlife Statute, 1996 (No. 14 of 1996), replaces most of the Game (Preservation and Control) Act except for the schedules of listed animals, and in part it is meant to provide for "sustainable management of wildlife." Wild animals are considered government property, but lawfully taken wildlife is the property of the permittee. The Act is enforced through the Uganda Wildlife Authority and makes provisions for the establishment and maintenance of wildlife protection areas and requires a comprehensive management plan for these areas.[30]

### European Union

The European Union (EU) has been implementing CITES since 1984. Two new regulations finalized in 1997 define the EU's wildlife trade legislation. They pertain to international and internal EU Member trade in wildlife. The Council Regulation (EC) No. 338/97 regulates trade in the species listed in the three CITES appendixes. The regulation deals with import and export permit requirements, allows seizures and confiscations, and allows appropriate penalties for infringement of the regulations. Commission Regulation (EC) No. 939/37 further details rules for EU Member States on the implementation of 338/97. Individual Member States of the EU are then responsible for passing laws to enforce these regulations.[28] National legislation for the 15 EU countries can be found at the European Commission website.[29]

### United Kingdom of Great Britain and Northern Ireland

The U.K. became a signatory to CITES in 1976. The Protection of Animals Acts of 1911, amended in 2000, covers cruelty and unnecessary suffering to domestic and captive animals, including elephants. The Performing Animals (Regulation) Act of 1925 requires trainers and exhibitors to register with a local authority. The Dangerous Wild Animals Act of 1976 requires that dangerous wild animals be kept so that there is no risk to the public and the welfare of the animals is safeguarded. The Zoo Licensing Act of 1981 requires zoo licensing and inspection. Administration of the Act is the responsibility of local authorities (Department for Environment, Food and Rural Affairs).[24]

Zoo Forum is an independent advisory body of the government established in 1999 to ensure that "captive wild animals are properly cared for." The Forum consists of professionals from the zoo industry, animal welfare organizations, research, and education; veterinarians; and others (Department for Environment, Food and Rural Affairs 2005).[24]

### United States

The United States became a signatory to CITES in 1975. The U.S. Endangered Species Act was passed in part to implement CITES and other international treaties.[27]

The Federal Animal Welfare Act (AWA) became U.S. public law in 1966 and was initially meant to protect dogs and cats and other animals in research. The enforcement of the law is under the jurisdiction of the U.S. Department of Agriculture (USDA) with authority delegated to the Animal Care (AC) program through the Animal and Plant Health Inspection Service (APHIS). In 1970, the Act was amended to include animal exhibitors. This covers the majority of elephant owners in the U.S.[5] The AWA requires that minimum standards of care be provided for most warm-blooded animals bred for commercial sale, used in research, transported commercially, or exhibited to the public. This includes animals exhibited in zoos, circuses, and marine mammal facilities as well as pets transported on commercial airlines. The law was again amended in 1976, 1985, and 1990. The regulations to implement the Act specify how the AWA will be enforced and include licensing and registration requirements and minimal standards of care. Animal Welfare Regulations are divided into Regulations and Standards. The regulations (Part 2) include handling, veterinary care, and record keeping requirements. The standards (Part 3) that pertain to elephants include housing, husbandry, employees, and transportation requirements.[59]

With the increasing threat to the survival of the Asian elephant, the Asian Elephant Conservation Act was written to assist and support in the conservation of Asian elephants by providing financial resources for conservation programs of Asian elephant range nations. The Act provides for a special fund and gives the Secretary of the Interior authority to approve projects using these funds. The Act prohibits funding from being used for captive breeding except for elephants to be released into the wild. An advisory group made up of individuals from public and private organizations assists in implementing the Act.[8]

The African Elephant Conservation Act (1988) is similar to the Asian Elephant Conservation Act. It supports conservation efforts in African countries and prohibits the importation of raw or worked ivory that originates from countries that don't meet the provisions of the Act.[2]

### Canada

Canada became a signatory to CITES in 1975. The Wild Animal and Plant Protection and Regulation of International and Interprovincial Trade Act (WAPPRIITA) was passed in 1992, became effective in 1996, and implements the CITES agreement in Canada.[18] There are penalties for animal cruelty violations at the federal level, but other animal welfare–related legislation is under the jurisdiction of individual provinces. Many provinces require a license to possess captive wildlife. In general, animals are seen as property, and charges against the owners are considered as property charges. Canadian law has remained essentially the same on this issue since 1892.[44]

### Australia

Australia became a signatory to CITES in 1976. There is a nonstatutory National Consultative Committee for Animal Welfare. The Committee, established by the Minister for Primary Industries and Energy in 1989, functions to help states and territories keep their animal welfare laws uniform and to monitor and advise on welfare issues. The Committee consists of government and industry officials, representatives of states and territories, and animal welfare groups (Australian Government Department of Agriculture, Fisheries and Forestry). The Wildlife Protection Act (1982)[61] implements Australia's obligations under CITES. Zoos are required to follow the laws of the individual states and territories, and these laws are linked to a Code of Practice written by the Australian Regional Association of Zoological Parks and Aquaria.[12]

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# 4 Behavior and Social Life

Bruce A. Schulte

## INTELLIGENCE IN ELEPHANTS

A fundamental, universal concept of animal psychology defines intelligence as the ability to solve problems.[108] Whereas for humans the term intelligence evokes ideas of encyclopedic knowledge, IQ tests, and grades, for nonhuman animals, such ideas are irrelevant. Attempting to place animals on some linear scale of intelligence is not only fruitless but detracts from our ability to understand the animal in question. The existence of animals in a wide variety of environments suggests that all animals have solved problems of survival and reproduction. However, the human concept of intelligence tends to focus more on the ability to solve novel problems, rather than those selected for over evolutionary time. Thus, intelligent behavior reflects the ability to learn and deal with new situations.[81] We might view the ability of elephants to dismantle an electric fence by pushing a tree onto it, while cattle remain inside the fence, as a sign of greater intelligence by elephants. Yet, pushing down trees is a natural part of elephant but not bovine behavior.

In terms of testing for intelligence using an experimentally valid approach, relatively little work has been done with elephants. Elephants are long-lived, traverse a broad range of habitats over a lifetime, and possess a proportionally large and complex brain that goes through the majority of its development after birth. All are qualities often associated with the ability to solve problems (Rogers 1997, Wynne 2001). Tool use and manufacture may be associated with higher cognitive aptitude and the ability to solve problems creatively. Elephants use a wide variety of tools in wild and captive settings,[11] primarily to reduce ectoparasites, to facilitate thermoregulation, and to obtain and manipulate food items before consumption. Through the secondary use of the dexterous and powerful trunk, they may strip bark, knock sod off grass clumps, or delicately choose selected leaves from among the thorns of acacia trees; the use of a branch to swat flies is a relatively facile extension of their behavioral repertoire.[28]

Consciousness (sometimes measured as self-awareness) is another potential correlate of intelligence. Povinelli[64] failed to show that elephants recognize themselves in a mirror, a standard test of self-awareness,[24] although the two captive Asian elephants were able to use a mirror to assist them in finding food hidden from direct view. Discrimination and memory tasks are additional potential measures of intelligence. Such behavior was examined through operant conditioning in a young Asian female at the Muenster Zoo by Rensch,[77,78] and the findings supported the idea that elephants are relatively rapid learners. Savage[84] tested three captive female African elephants using a two-choice object discrimination task. The females demonstrated learning over the study. A more recent study on Asian elephants in working camps of Burma failed to support extensive visual discrimination and memory using operant conditioning. The authors concluded that elephants did not show extraordinary intelligence,[49] although aspects of their experimental design may lead one to question this conclusion. Nevertheless, elephants are trainable as adults, have excellent retentive memories over time, and are adept at producing a wide range of behaviors appropriate to a broad array of situations. Practically, the understanding of these abilities strongly indicates that the safest way to interact with elephants would be to appreciate their potential for highly cognitive responses and their capability to resolve perceived risky situations.

## FREE-RANGING ELEPHANTS

### Family Structure and Learning

Asian and African elephants have a similar family structure based around a related group of females headed by the matriarch, who is typically the eldest female.[7,8,12,17,42,44,97] The family group generally consists of 8–12 individuals for the African savanna species (*Loxodonta africana*) and the Asian species (*Elephas maximus*), although smaller and larger groups are not uncommon.[18,32,45,47,97] The African forest species (*L. cyclo-*

*tis*) resides in smaller units, typically only a female and her current offspring.[101,102] For the remainder of this chapter, mention of African elephants refers only to the savanna species because so little is known about forest elephant behavior. African elephants sometimes gather in extended family units referred to as *closely related families,* namely, kin groups or bond groups;[12,45] assemblages of bond groups (*clans*) may gather at times, exchanging extensive greetings and interacting intensively.[12,14,32,44,45,97] Recently, Wittemyer[107] quantitatively analyzed the socioecology of savanna African elephants. It was shown that the fission-fusion nature of elephant society involved four tiers of social interactions (mother-calf units, family, bond group, and clan). The third and fourth tiers were responsive to ecological changes such as season, but the family unit was stable across season. Coalescence of unrelated individuals into a social unit is probably uncommon but not unknown[45] and may be more frequent after extreme alteration of family structure, such as through poaching (personal communication, Joyce Poole, Tufts, 1989). Asian elephants travel in family units, but bond and clan level aggregations apparently do not occur or are at least not common.[18,76] However, in both Asian and African elephant society, calves lie at the core of the elephant family with the matriarch serving as the head.

Within the social group, related females, especially young, nulliparous females are engaged as allomothers.[13,36] Allomothers may not only aid the learning of calves but may benefit from the experiences of caring for the young of another female. After several months, calves are exploring their world; their learning is facilitated greatly by the regular presence of their mother or a relative.[36,48] Older matriarchs especially facilitate learning as other herd members imitate their actions. For example, following playbacks of less familiar elephants, herds with older matriarchs were more likely to display investigative smelling behavior than herds with younger matriarchs.[40]

## Hierarchy Within and Between Family Groups and Among Males

In the female-led herds, age, size, kinship, and probably reproductive condition affect the status of an individual.[13,69] Because residency within the natal herd is typically lifelong, strong bonds are established and information is accumulated. As typically the oldest individuals in a herd, matriarchs are repositories of knowledge.[40] Although little explicit work has been conducted, groups with older matriarchs may have a competitive edge over other herds. Dublin[13] indicated calves of subdominant females suffered higher mortality than those of dominant females, attributed mostly to differences in rainfall and available forage for the lactating females. Variation across herds attributed to competition has not been documented, but in a study of African elephants, families led by older matriarchs (>35 years old) were larger than families led by younger matriarchs.[107]

Males disperse from the natal group as young teenagers.[59] They often assemble in bachelor groups[17,59] and spar frequently to assess their status.[44] The loss of older males in a population may select for an earlier age of dispersal, perhaps increasing the opportunities for mating.[54,96,97] Teenage male Asian elephants go through a moda musth in which the temporal gland secretion releases a sweet, honeylike odor.[70] Such a signal apparently indicates the low status of the sender, avoiding unnecessary conflict with older, more dominant males. Studies with a wild population in southern India indicated that younger males are less skilled at detecting the precise ovulatory status of females.[76] Young African elephants may exhibit a similar condition. As males reach ages 18–20, they are larger than females in stature and become viable candidates as mates.[17,32,42,56] The condition of musth occurs sporadically at first and then regularly on an annual basis.[16,27,38,56,60] Musth is similar in its signs, physiology, and behavior between Asian and African elephants[60] and to a lesser extent in the chemical nature of the secretions and excretions.[67,70,72] When in musth, males are dominant to nonmusth males and more likely to associate with females.[56,58,100] Like conspecific captive male Asian elephants, wild males detect phases of the estrous cycle in females, and it is suggested that the temporal extension of the preovulatory period effectively provides a synchrony between the sexes for successful reproduction.[76] This study demonstrated, in addition, that males in musth investigated preovulatory females and their urine more than nonmusth males, supporting the idea that musth males are reproductively dominant. In confined regions such as Addo Elephant National Park, South Africa, male conflict results in fatal combat more frequently than considered normal in unconfined populations.[106] When not in musth, the positively correlated attributes of size and age are the primary determinants of dominance. Slotow[91] demonstrated that older male African elephants could reduce the aggressive behavior of younger males in a confined population.

## Home Range

The area over which an elephant travels depends on the availability of resources, notably water, food, and mates. Because resources vary seasonally, so do the extent of elephant movements. Some methods for measuring home range and their implications for African elephant conservation are reviewed by Osborn.[52] Predation and human pressure also greatly influence habitat use by elephants,[4,104] perhaps to a greater extent today than any other single variable.[52] Female groups have overlapping ranges and may coordinate movements.[10,18] African and Asian elephants (at least Asian elephants in Sri Lanka) may differ in the tightness of suprafamilial associations. In a study of a disturbed population in Zimbabwe, family groups showed coordinated movements over space and time, but a matrilineal relationship was

not evinced through genetic analysis.[10] Genetic affinity is suspected for the Amboseli population. However, in Sri Lanka, associations between family groups were not observed, suggesting that clans may not form.[18] An earlier study in southern India[4] suggested that overlap by family groups may indicate a clan status of organization, but Fernando and Lande[18] question a level of organization above family groups for Asian elephants. Thus, in African elephants, ranging behavior may be influenced to a greater degree by the behavior of extended kin compared to Asian elephants.

Estimates of the actual amount of land used by an individual elephant over its lifetime vary widely, perhaps a reflection of the difficulties in obtaining such long-term data.[99] Values for both species show a low of 34 $km^2$ to a high of 6,400 $km^2$ with similar numbers for males and females. Although males and females may use habitat differently, exclusive regions for one sex are not evident. Even in the same region over a similar period, elephants can use vastly different amounts of land. Rainfall can be a strong determinant of home range size, but primarily in populations where water availability can be limiting. Males may be less restrained by water availability because of their large size and solitary nature.[94] Water also affects the amount and quality of forage, which further influences elephant movements. Commonly associated with rainfall and forage is reproductive receptivity; hence, the availability of mating opportunities also can affect elephant movements and congregation patterns.[14]

### Antipredator and Foraging Behaviors

Because of their large size and gregarious nature, matriarchal groups of elephants are relatively free of predation. Like precocial ungulates, newborn elephant calves are able to walk within hours and can soon keep up with normal herd movements. Still, young elephants are vulnerable to predators, especially if they become separated from the herd.[107] Elephants actively thwart potential predators, such as chasing lions from a water hole. Yet, in general, elephants are not very vigilant; that is, they do not spend much of their time on the lookout for predators.[9] Healthy adult males are formidable opponents and typically escape predation by their size and ability to defend. Male behavior is less likely to be affected by predators than that of females. Sick, injured, or aged elephants are more susceptible, although elephants have been observed to help those in such distress. Still, unlike most herbivores, predation pressure, except possibly human hunting, is probably not a major selective factor on foraging decisions by elephants.

### Foraging

Elephants live in a variety of habitats; thus, their diets are broad, incorporating grasses, forbs, fruits, bark, leaves, twigs, and roots.[99] The combination of a manipulative trunk, resilient tongue. and powerful body permits elephants to feed on small plants to large, thorny trees. Although elephants often take plants in accordance with their relative abundance, they do select for and against some species.[25,83] The ability to forage so widely is refined over development. Initially, calves feed only on milk, gradually experimenting with forage and regularly consuming such at several months old.[99] Suckling continues for the first 2 years, although opportunistic suckling can continue for much longer.

The size dimorphism in elephants through development and between the sexes suggests that type and location of forage might vary with age and sex. In a study at Chobe National Park, Botswana, male African elephants were less selective feeders than were female family units.[93] When in groups, larger females browsed higher in the canopy than smaller, younger elephants; however, adult males feeding alone did not feed higher than females and their young.[95] Overall, males fed in one place on more parts of the plants for longer, and females browsed a wider diversity of plants to a lesser extent. Males appeared to require a quantity of vegetation, and females were more selective of quality. To some degree, this contrasts with crop raiding behavior. Asian and African male elephants are more likely than female groups to raid crops, even forming small, temporary consorts of males for the raid.[30,53,100] Furthermore, males selectively choose harvested, bundled rice over ripe, standing crops.[68] As with most polygynous mammals, males have a higher reproductive variance than females; crop ingestion may increase nutrition and subsequent growth, allowing males to better compete for reproductively active females.

## REPRODUCTIVE BEHAVIOR

Males and females mate with more than one individual during a given estrus, but females usually have only one offspring per pregnancy. Because only one male can be the sire, the mating system is polygynous.[58] Barnes[3] suggested that males might exhibit female defense polygyny under certain ecological conditions, but typically, males defend only individual females during brief receptive periods.[57,58,83,96]

Adult male and female elephants live in very different social structures, not unlike that of other ungulate species where the sexes are highly independent except during periods of mating.[65] Females provide sole care for offspring of each sex during their 10–15-year development until sexual maturity (females) or dispersal (males).[13,14] Age of first ovulation, based on a 22-month gestation period, ranges from 7–23 years, but typically occurs in the early teenage years for wild females.[13,97] Females generally have their first calf between 10–16 years of age.[46,105] Although males are capable of sperm production at a similar age,[16,35] social maturity in males occurs later than for females, and males are not likely to mate until their late teens or twenties.

Male and female elephants signal their reproductive readiness through postural, auditory, and chemical signals. Although males do not have to be in musth to mate, musth males appear to be preferred by females.[58] The body posture of a male in musth is distinct,[56] and aggressive intent is readily apparent by the raised head, extended ears, and forward motion.[42] Moss[44] described behaviors indicative of estrus in African elephants, including enhanced wariness, spatial separation from the herd, leading males about at a walk or chase, and consorting with a single male for several days. Because infrasonic vocalizations may carry over many kilometers, females may advertise their reproductive status to attract males.[34,62] Females also may vocalize following a mating.

Upon entering a herd, males inspect the genitals, fresh feces, and urine deposits of each female, even very young ones. Males display a flehmen response to females approaching ovulation in which the trunk tip transfers material from the source to the ductal openings of the vomeronasal organ in the roof of the mouth.[71] Rasmussen and colleagues[73] identified a compound in the urine of female Asian elephants, (*Z*)-7-dodecen-1-yl acetate, that serves as an estrus pheromone. The concentration of this compound increases in the weeks preceding ovulation and then ceases to be released postovulation. Although this compound does not occur in African elephant urine, a similar signal appears to occur in preovulatory African elephant urine.[2] African males exhibit very similar inspection behaviors as Asian male elephants.[26,61]

Mating is a relatively brief act, requiring the female to stand while the male mounts and copulates. The male may use his trunk to position or hold the female, but her cooperation appears necessary for successful intromission. Mounting is practiced not just by reproductively active males, but also by younger elephants, perhaps as a sign of dominance or as a means of learning.

## PLAY BEHAVIOR

Animal play defies a simple definition but may function to prepare individuals for future unexpected or potentially stressful events.[92] Three major forms of play are object, locomotor, and social.[15] Although young elephants have been observed to carry sticks, run about, chase other species and caper with conspecifics, there has been relatively little work conducted on elephant play. The only in-depth study, on African elephants in Amboseli, showed that males played more aggressively than females, often staging mock fights and typically interacting with same-age males, including unfamiliar males.[36] Young female African elephants tended to engage in more subdued, social play, generally with younger members of the same herd.

Recent and ongoing studies by the author's group (e.g., Bagley,[2] Loizi,[37] Schulte and colleagues,[89,90]) are focusing on the relationship of play behavior to the sexually dimorphic development of communication patterns in African elephants.

## COMMUNICATION

Elephants are quite adept at receiving vibrations through the air and possibly even through the ground.[50] Audible vocalizations have been characterized for some time (e.g., McKay 1973, Berg 1983), and the low pitch of some sounds is below human hearing (infrasound). Payne and colleagues[55] described these sounds and further work by Poole and colleagues[62] and Langbauer and colleagues[33] supported the hypothesis that infrasound could serve to communicate between elephants one to several kilometers apart. Female African elephants can recognize the calls of family or bond group members, although the audible frequencies of the call may be the more important component for communicating social identity.[41] Elephants also appear capable of modifying their calls based on experience.[63] In this study, captive African elephants (one in Switzerland, the other in Kenya) imitated the chirping of an Asian elephant and trucks, respectively. The authors attribute this ability to the selective value of learning sounds that are individual-specific to maintain cohesive bonds after periods of separation. Overall, females have a greater repertoire of calls than males,[59] which is likely related to the extensive social network experienced by females over their lifetime.[39]

See also Chapter 32.

## CAPTIVE ELEPHANTS

Nearly one-third of the Asian elephant population resides in captivity, mostly in range states, where elephants traditionally were used in lumber camps and other such enterprises. Approximately 1,000 Asian elephants are housed in zoos and circuses worldwide, similar to the number of African elephants held in captivity.[99] The discussion below focuses on elephants living in primarily western world facilities.

### Social Grouping

In North America, elephants in captivity are maintained in a social structure that reflects some of the basic attributes of the wild situation but with some pointed differences.[86] Females reside in groups of two or more, although typically not in known, related family units. Because the birth of elephants is not common in captivity, calves are not a main component of most groups. The projection is that elephants will not be maintained in captivity through breeding.[29,43,51,103] Sukumar[99] reports that, historically, captive populations have never been self-sustaining. Yet, elephants in captivity can make a valuable contribution to human education and elephant conservation.[80]

Although true matriarchs rarely exist in captivity because of the group structure, matrilineal dominance still is evident and commonly determined by size and temperament.[20] Pubescent and adult males often are separated from females and each other, although visual, audible, and olfactory contact is available. As summarized by Schulte,[88] captive social structure differs from the wild in that groups are smaller, composed of mostly unrelated individuals, calves and males are rare, and some individuals live in mixed species (African and Asian) groups.

### Maintaining African and Asian Elephants Together

Because of the desire to breed elephants in a more natural social setting, the housing of the two species together is becoming less common.[82] The possibility of disease transfer between the species, especially herpesvirus,[19,79] also motivates managers to keep the species separate, but there is no evidence that the species cannot be housed together amiably.

### Special Challenges of Socialization in Elephant Groups

Although elephants in captivity do not generally reflect a kin-based social structure, they display many of the same behaviors as wild elephants. For example, the production and response to chemical signals[74,76,87] and to vocalizations[33,34] are similar in captivity and the wild. As in the wild, social dominance is achieved by a combination of size, age, temperament, and experience.[1,88]

Humans desire animals that are tractable and healthy in a captive situation, with regular human presence and typically some level of direct interaction if not contact. The challenge is for wild animals to develop a relatively normal behavioral repertoire and set of social skills while living within the boundaries of a human-established environment. To some extent, wild elephants in many locales are faced with the same issue, except that the spatial boundaries are larger and the types of interactions can be more varied and even severe. For captive elephants, the better arrangements are likely to be those with family units appropriate in size and composition to the species and with adequate space and a varied environment to maximize physical and mental welfare.[98] The mingling of tame and wild elephants in India provides a rich environment for the working elephant, but this situation is not available to elephants outside of range states.[23,99] In light of the social structure of wild elephants, maintaining solitary elephants, even males, may not be the best condition. However, even in this situation, the individual history of the elephant, its health, and the nature of its current housing situation should be considered carefully before increasing the size of its herd.

The adjustment of the elephants to the new facility, routine, and resident elephants is of primary concern after a successful transport. Scientific studies on the process of introducing elephants into new groups are rare, because most introductions have relied on the expertise of the personnel involved. In a study at the Muenster Zoo, three female Asian elephants were introduced to five resident conspecifics. Although all elephants showed changes in behavior following the introduction, after several months, behavioral and cortisol levels were similar to preintroduction, indicating that the introduction was not overly stressful.[85] Although the value of experience and expertise of personnel should not be underestimated, creating a protocol for introductions and documenting the process in a scientifically valid manner can have tremendous benefit to all people involved with captive elephants. Burks and colleagues[6] suggest a sequential method of introduction that is composed of four main steps: 1) document baseline behaviors and hormones of all elephants involved; 2) permit noncontact sensory (visual, auditory, and olfactory) exposure; 3) allow limited physical contact; and 4) complete the physical introduction.

### Behavioral Repertoires

The most extensive cataloging of elephant terms, including behaviors, has been compiled by Kahl and Santiapillai.[31] No single standardized ethogram or behavioral catalog for elephants exists, and the number of behaviors depends largely on the specific research questions.[90] Our research group uses a state and an event behavioral catalog to pursue our interests in chemical signaling and development (Tables 4.1 and 4.2). Schulte and colleagues[88] describe a few aggressive or dominance-related behaviors by captive female African elephants in a study on episodic acyclicity. Additional behaviors such as swaying, weaving, head bobbing, and trunk tossing are often considered in studies on elephant enrichment.[21,22]

### Detecting Aggression or Illness

By understanding the behaviors that elephants use to communicate with each other, humans may be better prepared to comprehend the messages elephants send to them. Threat or fear behaviors indicate that the elephant is uneasy and the consequences for a nearby person could be dangerous. Although an understanding of typical signs is extremely valuable, only through watching elephants and understanding the traits of each individual can one fully discern changes in behavior that might be indicative of aggression or illness. Animal caretakers, including managers and veterinarians, should take the time to watch their elephants.

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**Table 4.1.** Common State Behaviors of Elephants

| State Behaviors* | Definition |
|---|---|
| Chase | One elephant pursues another, other flees |
| Defecate | Release feces |
| Stand | Remain in the same location for at least 2 seconds |
| Suckle | Nipple contacts separated by less than 30 seconds of time off nipple |
| Urinate | Release urine |
| Walk | Leave location while all four legs are moving in a steady pace |
| **Trunk Actions** | |
| Drink | Place water into the mouth usually with trunk, except for young calves |
| Eat | Take nutrients into the mouth |
| Rest trunk | Place approximately one-quarter of the lower trunk on the ground for at least 5 seconds |
| Object play | Use the trunk to manipulate an inanimate object or splash the tip of the trunk into water |
| Sparring | Entwine trunks/tusks and push against another |
| **Care** | |
| Bathe | Submerse most or all of body in water |
| Dust | Use the foot or trunk to place dirt particles on the body |
| Hose | Spray water on body |
| Lay | One side of the torso in contact with the ground |
| Mud | Use the trunk to throw mud particles on the body or moving body rapidly in a mud hole |
| Other | Behavior not defined in ethogram |
| Not visible | Elephant has moved out of sight |

*State behaviors have a measurable duration. As needed, some can be categorized as events.

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**Table 4.2.** Event Behavior Performed by Elephants in Studies Focused on Chemosensory Behavior

| Event Behaviors* | Definition |
|---|---|
| **Spatial** | |
| Avoidance | Fail to initiate or cease forward motion toward another elephant or sample |
| Circling | Move laterally around sample area or another elephant |
| Near | Elephant within one trunk length of sample with trunk in down, open position |
| Proximity | Elephant within one body length of sample |
| **Primary Chemosensory** | |
| Sniff | Nasal openings hover over ground or conspecific without contact |
| Check | Touch ground with tip of either finger |
| Place | Entire nasal opening is placed on ground or conspecific and held briefly |
| Flehmen | Tip of trunk touches substrate or conspecific then placed on the VNO ducts |
| **Additional Trunk** | |
| Blow | Air is expelled quickly from nasal openings of trunk; often audible with mucus visible |
| Dig | Use trunk tip or foot to displace substrate |
| Dust | Throw substrate on body using trunk |
| Periscope sniff | Raise trunk is raised to air above head and hold level for at least 2 seconds |
| Pinch | Two fingers of trunk pick up dirt around the sample |
| Suck | Same trunk position as Place accompanied by trunk contraction; usually audible |
| Trunk flick | Bottom one-quarter of trunk moves up and down rapidly |
| Wriggle | Trunk twists and then untwists once at a moderate pace (slower than trunk flick with more of trunk involved) |
| **Trunk Tip Contact** | |
| Anus | Anal region |
| Body | Torso or areas not listed |
| Feet | Area below ankle |
| Genital | Penis or vulva |
| Head | Forehead and superior point of head |
| Mammary glands | Nipple region of mammary gland |
| Mouth | Inside oral cavity |
| Palatal pit | Contact to numerous, small blind-ended crypts located bilaterally dorsal, anterior mouth cavity |
| Tail | Contact to posterior appendage |
| Temporal gland | Contact to temporal gland orifice |
| Trunk | Portion of trunk starting from mouth area down to tip |
| **Body Contact** | |
| Back into | Rear end moves backward into another individual |
| Body rub | Torso-to-torso contact |
| Head butt | Quickly using the head to make contact with the body of another individual |
| Kick | Use legs to strike at another |
| Lean | Put body weight on another elephant |
| Mount | Stand on hind legs, forelegs resting on body of a standing elephant |
| Present | Turn backside toward another |
| Push | Use the body to displace another elephant from its location |
| Roll | One elephant is on the ground; other elephant is on top |
| Slap | Strike with trunk |
| Trunk on head/back | Place the entire length of the trunk on the head/back and hold position for at least 2 seconds |
| Trunk wrap | Entwine trunks, often accompanied by pulling or constricting |
| Tusk | Strike another elephant with tusk |
| **Other Behaviors** | |
| Ear motion | As needed, various actions of ear can be specified |
| Head shake | Head held above shoulders and moved side to side rapidly |
| Motionless | Body and appendages still for 5 seconds; note head and ear position |
| Penis erection | Penis fully extended and rigid |
| Penis peek | Only tip of penis visible |
| Penis pull | Trunk touches and pulls on penis |
| Penis slap | Penis struck against underbelly |
| Tail flick | End hairs of tail tip are dragged across the clitoris and then waved behind like a flag |
| Tail out | Tail out horizontally |
| Vocalize | Audible sound production; various types can be specified |
| Other | Behavior not specified in ethogram |

*Chemosensory definitions derived from Schulte and Rasmussen 1999, Bagley 2004, Loizi 2004, and Rasmussen 2005.

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# 5 Husbandry

John Lehnhardt

## INTRODUCTION

This chapter reflects the accumulated knowledge from the captive elephant management field. However, it is also expressive of the author's experiences and opinions accumulated over 29 years of studying, working with, and managing elephants.

The husbandry of elephants in the care of humans in zoological facilities continues to evolve as more is learned about their natural history, and as increasingly effective management techniques are developed. Thus, any husbandry chapter reflects only current knowledge and practices and cannot be considered as the ultimate dissertation on captive elephant management.

There are two recent and excellent primary resources for more extensive information related to elephant husbandry than may be provided in this brief treatise. One is the Elephant Husbandry Resource Guide.[18] The second is the curriculum of the American Zoo and Aquarium Association's (AZA) Principles of Elephant Management (PEM) course, which outlines the components of an effective elephant management program. Both provide in-depth treatment of many of the concepts mentioned here. It is strongly recommended that all those caring for elephants acquaint themselves with both works. When thinking of agriculture and farming, caring for and nurturing the land, plants, water, and animals to produce a valuable and sustainable product comes to mind. This chapter is based on that premise.

Husbandry is a subset of overall management. The focus of this chapter will be the aspect of husbandry that contributes to the overall effective management of elephants in the care of humans. Some of this treatise on husbandry will attempt to separate what we do know and what we do not know about elephants and provide some myth busting around some of the more controversial current aspects of elephant husbandry and management.

Elephant husbandry is not high technology. However, elephants are complex animals that people identify with and may come to care about passionately. Elephants have had a long relationship with man, and probably because of this long standing connection between our species and elephants there has been a great deal of controversy regarding how elephants are managed, both in the wild and in the care of humans. What elephants use and require in their natural environment and what that means for them and their managers in the care of humans is still quite debatable.[9] In general, there is a lack of strong scientific evidence to support what is truly the optimal approach to effective elephant care, including husbandry.[11] Many published statements are based on no science, poor science, or pure emotional responses. So-called "experts," who, in reality, have very little knowledge or understanding of elephants, publish a great deal of misinformation daily about elephants in the wild and in the care of humans. Even good science related to the elephant management field can, and is, used inappropriately. One would hope that everyone has the best interests of the elephants in mind, even when they disagree on management practices (Hutchins, in press). Unfortunately, much of the information published is meant to promote agendas, either to attack or to defend elephant management practices.

In this chapter, the focus will be on what has worked in elephant husbandry in the care of humans. It is hoped that the information will be used by elephant managers to improve their husbandry skills and that elephants will benefit from this treatise.

Asian and African elephants are somewhat different in their natural history, physical expression, and social organization.[23] However, there are many similarities between the species and, in general, most statements in this chapter will refer to both species, unless specifically noted that it applies to one or the other.

## COMPONENTS OF AN EFFECTIVE ELEPHANT HUSBANDRY PROGRAM

An effective elephant husbandry program has many components, each contributing to the overall welfare of

the elephants. Good husbandry is primarily preventive. The husbandry goal should always be to provide an overall environmental experience that meets the species' needs, contributes to the wellness of the animal, and yet provides safe containment and management options to deal with the species capabilities.

The most important aspect of any species husbandry program is an understanding of the species' needs from a physiological, social, and psychological context.

## Elephant Physiological Needs

**Diet.** Food and water are clear physiological needs that are sometimes taken for granted. However, proper diet and nutrition are basic to good husbandry. See Chapter 6 for more details. In the wild, elephants forage for many hours daily and have been known to consume more than 100 different species of plants.[23] They are poor digesters and rely on a strategy of eating large quantities of food and passing it through their digestive system rapidly.[23]

Overfeeding may be one of the most serious problems for elephants in the care of humans in European and North American zoological institutions. A veterinary colleague who has participated in surgeries and necropsies of wild range state elephants and those in the care of humans relates that fat levels in the wild state animals are virtually nonexistent, but elephants in human care in North American institutions and in private hands generally seem to have significant fat deposits throughout the body (personal communication, Dr. Mark Stetter, Orlando, Florida, 2004).

The development of excess weight on elephants in a nonrange environment is fairly easy to understand. Elephants in the wild go through times of nutritional stress during regular patterns of reduced rainfall or more severe circumstances of drought.[8] Much as caribou and reindeer store fat to survive the winter, elephants store fat to survive the dry season. In our institutions, we provide a relatively consistent food source year-round and would never think of stressing our elephants with food reduction to mimic what happens in their natural environment. Anyone who did might be considered cruel and inhumane. However, this dietary approach does not allow the elephants the opportunity to use up their fat reserves on a regular basis, and thus, they put on and retain excess fat. So, unless we provide a diet that is better balanced from a caloric perspective, we will continue to have elephants carrying excess fat deposits.

Excess fat may have significant impact on the overall health and well being of elephants in the care of humans, as has been shown for other species. Problems with conception and parturition, as well as leg and foot problems, could be impacted by excessive weight. Fat builds up when more calories are consumed than are expended, so caloric intake must be reduced or energy expenditure increased for an elephant to lose weight.

Dietary caloric reduction may be achieved in a number of ways. First, the overall diet should be assessed for caloric and nutrient content by a professional nutritionist (personal communication, Eduardo Valdes, Orlando, Florida, November 2003). The use of nutritional specialists has increased in North American zoological institutions in recent years as the importance of this field has been recognized for all animals in the care of humans. The elephants should be weighed regularly and appropriate target weights (see the section titled "Exercise" below) should be established for each animal, depending on age, reproductive status, and any other overall health parameters relevant to the individual animal. Weighing should be done yearly, at the absolute minimum, with monthly weighing highly recommended.

Grain supplements are often fed with little regard for their caloric impact on the diet. There are many products on the market and elephant managers all have their preferences, as do the elephants. The key is to feed the minimum needed for the individual elephant and situation to provide the required nutrient value, and no more.

Hay species, which makes up the majority of the elephant diet in most Western institutions, can also make a big difference in the caloric intake for the elephants. Regular assessment of the hay nutrient value is necessary to formulate an appropriate feeding regimen. Generally, lower nutrient quality hay may provide a better overall diet than a high nutrient quality product. Specific diet components are covered in detail in Chapter 6.

The optimal nutritional regimen for elephants is still under investigation. However, from a husbandry standpoint, delivery of the desired components of the diet is important. First, variety is critical, with mixed fresh browse species provided daily, if possible. Browse may require greater manipulation to ingest and thus provides both increased activity and enrichment. Supplemental browse must be included in the calculation of the caloric content of the diet. Low nutrient quality browse may be effectively used to offset higher quality hay and grain products to produce the proper overall diet nutrient balance (personal communication, Eduardo Valdes, Orlando, Florida, November 2003).

Hay of appropriate species, if this is the major nutritional component, should be provided for a majority of the day and night, because elephants in the wild have been reported to spend as much as 50–75% of their daily activity budget eating.

Providing food in a controlled environment to simulate the natural feeding patterns requires creativity. Some obvious aspects of food presentation should be noted. Elephants in confined areas, such as night stalls or holding enclosures, soil their food readily. Feeding stations should be located in the area least likely to be soiled by manure and urine. If food is directly placed on the flooring, it should be at the top of the slope of the floor, and the flooring material (such as concrete)

should be easily cleaned. Elevation of the food off the floor is ideal as it reduces spoilage.

In the wild, elephants generally do not have access to water at all times, and the water available certainly may not be what would be considered clean and potable. African elephants in some areas are reported to drink only every other day.[15]

Most institutions provide clean, potable water for their elephants at all times. In fact, it is a USDA-APHIS requirement in the United States that elephants be provided clean, potable water. There are several ways to accomplish this and many creative solutions to prevent the elephants from contaminating the water with food or feces. The water supply should always be elevated at least 0.6 m (2 ft) off the floor. Continually running water is wasteful, costly, and not environmentally friendly. Elevated water basins with level-limiting floats work well. They should have a minimum volume of 7.6 l (2 gal) for one trunkful of water, and be able to refill in about 30 seconds. Positioning of the basins outside the enclosure within trunk reach effectively prevents soiling. These basins should be designed to drain easily and should be cleaned and disinfected regularly.

## Feed Storage

When at all possible, elephant hay or grass should be stored in a closed environment on slatted pallets placed on a solid floor. This will reduce rodent infestation and allow for air circulation around the food source. Grain or supplements should be stored in unopened bags on pallets above a solid floor or in metal containers or bins to prevent spoilage from moisture or infestation from pests. Feed items should be organized to allow stock rotation to prevent spoilage due to prolonged storage. The primary storage facility should be centralized and the diets delivered to the elephant facility as needed. This reduces waste and allows better inventory control. Usually a 1- or 2-day supply of food at the elephant barn is sufficient.

**Handling fecal and urinary waste.** Handling significant quantities of urine and feces are daily tasks for elephant holding facilities. Elephants can produce in excess of 68 kg (150 lbs) of manure per day per animal. African elephants produce larger amounts than Asian elephants, and mature bulls may produce twice the amount of a mature cow. African elephants have a tendency to have less form and more volume to their feces. African elephants also throw their feces with regularity and even like to roll in it. Thus, cleaning after African elephants requires significantly more effort.

Elephant solid waste has been composted in many institutions and even sold as fertilizer (ZOODOO®). Most manure is removed the old-fashioned way, by shovel and wheelbarrow, placed in a dumpster, and removed to landfill. A few institutions with large elephant herds are using trough augers to transport the manure from the barn stalls directly into the dumpster, saving significantly on labor.

Stalls should be cleaned every day of all debris, hosed, scrubbed, and periodically disinfected. Drainage is important to prevent urine buildup where the elephants stand and lie down. It is preferable for drains to be positioned outside the stalls and the slope toward the drain should be about 0.32 cm per 30 cm (1/8 inch per ft). Large drains with good screening are required to prevent hay blockage while cleaning.

**Exercise.** The other aspect of calorie management is exercise and activity levels. The more active an elephant is, the more calories it burns. Thus, to maintain an elephant at an appropriate weight, the caloric expenditure must equal the caloric intake. We don't have any measurement of how much exercise elephants need. However, after a specific weight range is established as appropriate for an individual animal, if the animal stays within that weight range, we can at least assume the diet and exercise levels are balanced. Increased exercise levels would require increased caloric intake to maintain weight, and decreased exercise levels would require decreased caloric levels.

Activity levels may be increased in many ways. It may be assumed that primary energy expenditure would be associated with walking, trunk manipulation, and ear movement, in that order. Assuming that walking is the primary energy user, stimulating walking should be the primary method of providing exercise. The food should be distributed throughout the enclosure to stimulate foraging activity and walking.[21,22] Food may be hung in rope baskets above the floor. Other specialized feeders have been developed in many institutions that stimulate increased activity to acquire the food. Elevating the food also reduces urine and fecal contamination. This is particularly important when elephants are stalled in restricted areas for significant portions of the day or night.

Training programs also provide exercise and stimulation that promote movement and may contribute to overall well being. Just from a weight perspective, more activity burns more calories. Every management system may develop a training regimen that increases the elephant's activity level and virtually anything that accomplishes this may be useful.

Increased herd sizes and age distributions also stimulate activity. Calves are great calorie use stimulators, both from a consumption basis for the mother by the calf's nursing and from the activity levels the calves generate in the herd. In the wild the calves are regularly cared for by younger, nonreproductive females, or allomothers. This probably allows the mothers to focus on getting sufficient calories to nurse the calves without spending too much energy looking after them.[15]

Because weight management appears to be an important aspect of elephant overall health assessment,

weighing elephants on a regular basis is an important part of elephant husbandry. Every elephant facility should have the capacity to weigh its animals. Scales can be built into restraint devices or into corridors. Portable truck scales are readily available on the market. Local highway patrol units usually have portable scales they may be willing to provide for periodic use. The scales should have a minimum 3175 kg (5 tons) capacity for elephant cows and 7 tons for adult bulls. Smaller portable platform scales may be used for younger animals, particularly newborns whose weight should be more accurately measured.

## Enclosure Sizes

A great deal of discussion has occurred recently regarding appropriate indoor and outdoor enclosure sizes for captive elephants; however, there is no published scientific data that delineates optimal enclosure size for health and welfare. This particular lack of knowledge leads to significant controversy over how much space elephants need when in captivity. It is not known how much space is necessary for optimal well being.

Critics of elephant care institutions often promote a myth about elephants related to how much they normally travel, stating that elephants travel as much as 80 km (50 miles) in a day. This is frequently used as an argument to conclude that no facility can provide sufficient space to accommodate an elephant, and therefore, elephants shouldn't be kept anywhere but in the wild.

Unfortunately, we don't really know how far wild elephants travel in an average day. There is no published scientific paper that has studied this for even an isolated elephant group in the wild. There are certainly a few reports indicating that some elephants have, in fact, traveled that sort of distance in a day. There is also recent data from Africa indicating that some elephants have moved as little as 3.2 km (1–2 miles) in a day[15] (personal communication, Iian Douglas-Hamilton, Washington, D.C., April 2002). So, what is normal? How much exercise in terms of walking will supply sufficient exercise to meet an elephant's needs? The answer is not known. The issue of space and exercise should be looked at from a different perspective. We need to answer this question first: Why do elephants travel?

Elephants travel in the wild to find resources, whether it is for food or water, or social interaction. If resources are abundant and close by, elephants do not need to travel far, and they probably don't travel far. If resources are distant or scarce and widely scattered, elephants may move great distances. The amount of exercise an elephant needs is not a set figure, but rather dependent on resource availability. Because in most managed care situations, elephant food is provided on a regular basis in sufficient quantity, there is much less need for travel. So, determining a minimum amount of exercise cannot be based on what distances elephants need to travel in the wild. It must be determined by other parameters. Likewise, the minimum size of the enclosure needed to provide the appropriate amount of exercise cannot be quantified. It is not possible to use objective measurements for size of enclosures and exercise. Instead, we look at well-being parameters, such as reproductive success, muscle tone, lack of arthritis, and, most important, appropriate weight. In this case it is not the size of the enclosure, but how it is used by the elephants and how the design and manipulation of the various aspects of the enclosure are managed.

## Skin Care

In the wild, elephants care for their skin by bathing regularly, coating it with dust and mud, and rubbing off excess skin against trees and rocks. In managed care, the skin requires the same attention. This may be accomplished by providing environmental options for the elephants to do all this as if in the wild or by providing some of these options through human interaction. Diverse substrates in the yards and even indoors are needed to provide these options. Baths and body scrubs are done daily in many institutions to meet some of these needs. In colder climates with dry winter conditions, a lanolin shampoo may help maintain the skin condition and prevent cracking. See Figure 5.1.

## Foot Care

In the wild, elephants' footpads or calluses cushion their steps and protect their sensitive foot structures from punctures. Generally, they are thick and wrinkled on the bottom. In captivity, the condition of the feet and the amount of foot care needed may be measures of overall welfare.[7]

Captive elephants generally do not need a thick pad to protect them from environmental hazards. However, inadequate activity levels or substrate variety may allow footpads to overgrow. An effective husbandry program provides the proper substrate for adequate footpad wear and also monitors and manages pad care through an active foot-care program. This program must allow each foot to be inspected and cleaned daily, and the elephants must be trained to present their feet for nail filing and cuticle and pad trimming.

## Substrate

Elephants require varied substrates to provide for their physiological needs. Outdoor enclosures should have sand, clay, soil, and varied elevations. These allow elephants to wear down their foot pads, nails, and cuticles sufficiently to limit the need for foot and skin care.

Interior substrates are frequently concrete. However, a number of different substrates have been used in interior enclosures to both insulate and cushion the concrete for more comfortable standing and sleeping. Rubber mats, poured rubber floors, and even sand or natural substrates on top of the cement have been used. Unfortunately, no single interior substrate has proven to be

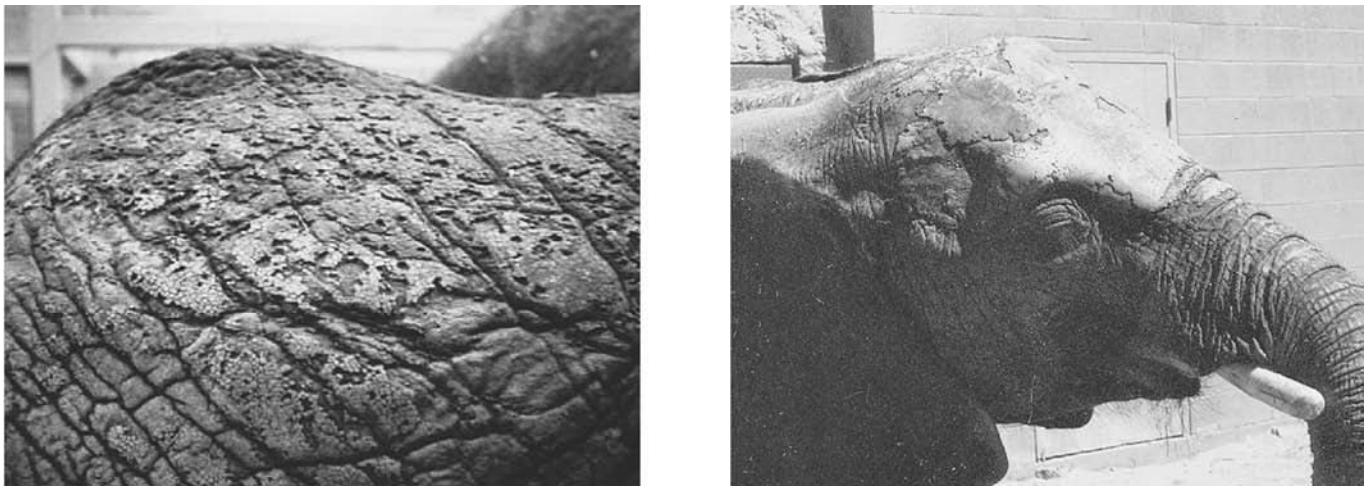

***Figure 5.1.*** *The consequences of poor management and lack of bathing are evident in the skin of this African elephant. The condition resolved with proper care after this elephant was transferred to another institution. (Photo courtesy of USDA, Animal and Plant Health Inspection Service.)*

completely adequate for all situations. Most poured rubber floors installed on top of concrete have limited life spans and wear down, or peel, under elephant pressure. Continued experimentation is needed to find effective floor padding for indoor stalls.

## ELEPHANT SOCIAL NEEDS

The most critical and often least considered aspect of elephant husbandry is the social structure and needs of elephants. Female elephants are family animals. They spend their entire life focused on rearing calves and interfacing with their family unit. Their involvement in calf rearing and care begins early in life, almost as soon as they are weaned, or when their mother has another calf. These young females often assist their mothers with calf care and provide allomothering for younger calves in the herd.[8] Because female elephants are known to remain reproductive throughout most of their lives, this is their primary activity beyond eating and drinking. Female elephants also appear to maintain lifelong relationships with their immediate relatives, particularly their female offspring.[15] The connection of elephant females in the wild to their family members and calves is well documented. Hence, the most critical aspect of husbandry and the focus of captive elephant management for female elephants should be the opportunity for reproduction, calf rearing, and maintenance of long-term familial relationships with their female relatives. Unfortunately, in North American captive management, this is the least frequently accomplished standard. Only 10% of North American female elephants currently live with their calves or a female relative. Long-term breeding plans recently developed by the AZA Elephant Taxon Advisory Group (TAG) Species Survival Plan (SSP) focus on encouraging long-term breeding and social grouping based on familial ties.[13,17]

Male elephants have very different social needs. In the wild, the males leave, or are driven out of, the family group as they approach sexual maturity and spend as much as 95% of their lives alone or in loose association with other bulls.[19] In the early years of adulthood, young bulls learn the capabilities of other bulls in their area and hierarchical social structures and status are established. As they age, grow larger, and compete effectively for reproductive opportunities, the bulls appear to spend their time eating and seeking out females. Elephant bull nature is competitive, rather than affiliative.[19] This causes many challenges for captive husbandry, because the bulls need little social interaction but definitely need competitive challenges to express their natural urges. Hence, the opportunity to breed is essential, but extensive social interface with other elephants is not.

Providing an appropriate social structure also contributes significantly to the well being of elephants in a number of husbandry-related areas. For example, for females and calves, the herd interactions provide the best environmental enrichment possible. Calves provide tremendous stimulation to the herd. The calves' elevated activity levels and social interactions increase the activity level of the entire group. As a result of having calves, particularly multiple, variant-age calves in the herd, all the elephants move around more, get more exercise, and put more wear on their footpads, potentially decreasing foot problems. The animals get more exercise, have better muscle tone, and are kept stimulated by looking after and interacting with the calves.

## ELEPHANT PSYCHOLOGICAL NEEDS

### Enrichment

With the largest brain on land and a complex social structure, elephants need an environment able to pro-

vide sufficient mental stimulation and environmental and behavioral enrichment to prevent lethargy, stereotypic behaviors, aggression to herdmates, and other behaviors thought to be inappropriate for elephants.

As described above, distribution of food throughout the environment and a complex social structure are the primary environmental stimulants that lead to psychological well being. In the wild, elephants make decisions constantly and need options in a managed environment so they can make choices about where to be, what elephants to be with, what to do, and when to do it. These options should include water for bathing, cooling, and play; mud and dust to provide for skin care, sun protection and insect deterrent; and manipulatives for play.

Depending on how natural an environment has been designed, additional enrichment items may be desirable. Scratching posts that allow for an elephant to rub all parts of the body may be constructed of tree trunks, I-beams, or concrete. Tree branches or irregularly shaped stumps may be pushed and explored by trunks.

## Containment

Another important aspect of a husbandry program is the complete understanding of the species' capabilities. Safe, secure housing and containment are essential for an effective captive elephant management program. There are as many different containment systems as there are elephant facilities. Containment systems are generally dictated by the requirements of the institution, the goals of the elephant program, and the management approach. For example, breeding institutions must provide for baby-proofing (keeping elephant calves contained) as well as for adult bulls in musth. This provides a much different structural need than for subadult or adult cows only.

Primary containment may generally be divided into several categories of structures: wet and dry moats, vertical bollards (vertical posts) with horizontal bars or cables, solid walls, chain or mesh grids, and electrical barriers. Often these different systems are used in conjunction. The primary purpose of the barriers is to keep elephants in. Secondarily, these barriers may need to keep people out or limit human access. The key for any containment structure is that it is strong enough to hold the elephants and is also safe and free of potential elements that may cause injury to the elephants. Moat containment can be effective for providing unencumbered viewing of the elephants in a zoological setting. However, historical moat designs frequently put an elephant's welfare at risk. Dry moats should have a drop-down of no more than 1.5 m (5 ft) and a soft bottom to provide for an elephant falling into or being pushed into the moat. A deeper moat with a hard bottom could potentially cause critical injury to an elephant. The moat must also have a width of at least 2.4 m (8 feet) to accommodate an elephant getting to its feet. The moat must also have a ramp or gate access point to allow the elephant to return to the primary enclosure without having to be hoisted with heavy equipment or a crane.

Wet moats used as containment must have a design and depth sufficient to prevent the elephants from climbing out over the barrier. Elephants are good swimmers and do float, but a containment depth of 2.7 m (9 ft) for Asian elephants and 3 m (10 ft) for African elephants at the containment edge should be sufficient.

Vertical bollard containment has become quite popular in many facilities. Steel bollards ranging in diameter from 10–30 cm (4–12 in) are anchored in concrete footers varying from 0.9–1.8 m (3–6 feet) in depth and 0.6–1.8 m (2–6 ft) wide. Generally, the gap between the bollards is about 35 cm (14 in). This allows for human access but contains elephants 2 years and older. Bollard heights range from 2.4–3.6 m (8–12 ft) and may be capped with a metal lintel connecting the bollards or concrete filled. Ideal height is probably 3.6 m (12 ft) to meet the needs of all species and sexes. Uncapped bollards should be somewhat large in diameter for strength. As bollard diameter increases, visibility into the enclosure decreases, so capping a diameter of 15.24 cm (6 in) probably is ideal for bollard structure.

Vertical posts with bars or cables have many varied installation methods. The posts may be wood or steel for cable barriers. Wood posts should be at least 30 cm (12 in) in diameter, with concrete footings, and spaced up to 2.4 m (8 ft) apart. Metal posts may be bollards structured as discussed for interior containment, or I-beams 15–30 cm (6–12 in) deep, set in concrete with the I of the beam set in the line of force from an elephant push. The height of the posts will depend on the proposed use. For standard containment, 2.4 m (8 ft) high is sufficient for any elephant. However, for adult bulls, the posts may need to be higher, about 3 m (10 ft), to carry higher cable strands—not for actual containment, but to limit the ability of large bulls to reach over the top. Electrified wires above the top cable will also deter reaching over. The cables should be braided steel at least 1.3 to 2.5 cm (1/2 to 1 in) thick, and can be either threaded through holes drilled in the posts or through D-rings attached to the posts. The cable may be electrified to discourage the elephants from pressing on them. The gaps between the cables need to vary based on the proposed use. For a breeding herd, the calf containment will require a maximum of 0.3 m (1 ft) gaps up to 1.2 m (4 ft) high, starting at 0.3 m (1 ft) off the base substrate.[1] Above the 1.2 m (4 ft) level, gaps of 0.5 m (20 in) to 0.6 m (2 ft) will work.

Horizontal bars instead of cables made of 7.6–10.2 cm (3–4 in) pipe or 5.1–7.6 cm (2–3 in) I-beams can also be used. However, the posts may need to be closer together with this application because the pipes can bend from head pressing or from elephants standing on them. Larger pipe or I-beams up to 15–20 cm (6–8 in) diameter should be used if the post separation is over 2.4 m (8 ft). Cable can be a less expensive solution.

Solid walls are best made of reinforced, poured concrete at least 20 cm (8 in) thick. When separating adult bull elephants, a 3.7 m (12 ft) high wall is needed for Asian elephants and a 4.3 m (14 ft) high wall is needed for Africans.

Galvanized steel mesh may be used to fill gaps between bollards or bars to ensure keeper or elephant safety and separation. These need to be at least 5 × 5 cm (2 × 2 in) woven and welded steel a minimum of 0.95 cm (3/8 in) thick. However, gaps over 0.6 m (2 ft) wide are susceptible to warping and breakage due to pressure from tusks.

Calf containment for breeding facilities requires special attention. Calves can slip through vertical gaps less than 25 cm (10 in), wide and all gates, doors, bollards and cable gaps must be measured to ensure calf safety. For a horizontal barrier, no gap greater than 30 cm (12 in) is secure. Below 1.2 m (4 ft), chains or cables treaded through D-rings may be added for gap closure. The challenge is to provide easily removable but secure areas for keeper access. This may be accomplished with short chains with end clips that can be easily taken on and off.[2]

## ROOF AND CEILING HEIGHT

Adult bull elephants can reach over 6.1 m (20 ft) off the ground, and are even known to stand on their hind legs in the wild to reach leaves or fruit. Thus a roof or shade structure in a facility holding an African bull should be at least 7.3 m (24 ft) in height. This also means that light fixtures, fans, cables, cameras, and any other items not elephant-proof must be at or above 7.3 m (24 ft). Any breakable item below that height needs strong steel mesh protection similar to the mesh described above. The manipulative nature of the trunk can unscrew bolts, and welding is suggested as the primary mode of affixing features elephants might interact with.

## KEEPER SAFETY ZONES

Keeper safety zones are areas near elephant containment where keepers can get out of the trunk reach of the elephants. The depth of the zones varies with the containment barrier structure and how the barrier limits the reach of the elephant. For standard bollard containment, with a 35 cm (14 in) gap between bollards, an elephant can reach its trunk a maximum of about 2.4 m (8 ft) through the bollards. So a keeper safety walk zone would need to be about 3.3 or 3.6 m (11 or 12 ft) of open space outside the bollards—2.4 m (8 ft) for the trunk reach and 0.9 to 1.2 m (3 to 4 ft) for the keeper walkway. However, for containment with different gaps than the 35.6 cm (14 in) bollard, or where elephants may be able to raise their head over the top of the containment, great care must be taken to measure the potential reach of the largest elephant to ensure a 0.9 to 1.2 m (3 to 4 ft) safety zone for the keepers beyond trunk reach.

## DOORS

There are many designs for elephant containment doors and systems for moving them. The key issues in door design are the strength of the door, how quickly it can be opened or closed, how much control the operator has of the door's movement, and how complete a view the operator has of the moving door. Whether the door is solid or provides visual or tactile contact with other elephants provides different structural challenges. A door with horizontal bars will provide an opportunity for the elephant to lift the door with its truck or head, requiring solid resistance from excessive movement. A door with vertical bars will need to resist side-to-side motion from the head and trunk. All doors, solid or not, need to resist direct pressure from an elephant's head from either side of the door.

Choosing whether to use hydraulic, pneumatic, electric, or manual door operators depends on the funds available and how the doors are to be used. Most door systems today are hydraulic, but there are a few manual designs that function well while providing good safety measures for the operator. The key for manual doors is that they still need to be operated from outside the elephant's reach and protect the operator from recoil if the elephant pushes the door during its movement.

Hydraulic doors should have animal-safe fluids made from either vegetable oil or nontoxic petroleum products. These are readily available. Any automatic door system should have the ability to move the doors manually, in case of a power failure, or have emergency power generation backup.

Many elephants have had their trunks damaged by being caught in closing doors, particularly hydraulic doors. It is critical that the full closing movement of any door be fully visible by the operator to ensure elephant safety. It is also highly recommended that protocols be in place to train elephants not to extend their trunks through closing doors and to not allow elephants to go through opening or closing doors. These behaviors are remarkably easy to train and provide a great safety benefit for the elephants.

## POOLS

Pools, indoors or outdoors, provide a source of enrichment, relief from heat in warm climates, and physical health for elephants. However, elephants do not spend significant amounts of their daily activity schedule in water in the wild. Hence, the pool should not consume an inordinate amount of the enclosure surface area. If a pool is not being used for containment, it does not need to be deep enough to submerge a standing elephant. A depth of 1.8–2.4 m (6–8 ft) is more than adequate. In fact, shallow water features for wading and water play may be more stimulating and better used than deep pools.

## ELEPHANT RESTRAINT AND RESTRAINT DEVICES—ERDS

Elephant restraint is an important part of elephant husbandry. Training elephants to accept restraint—either tethering, strapping, or in an ERD—allows safe and effective husbandry or medical procedures to be performed.

ERDs have become an integral part of elephant husbandry programs. There are many forms of ERD, including units that are stationary, those with movable walls, and others capable of 90° rotation. The purpose of the ERD is to confine the elephant to allow for care procedures. Elephants should be acclimated to restraints on a regular basis. They should be walked into the ERD consistently to allow lowered stress use of the device when needed.

In North America, ERDs have become quite complicated and mechanized. Traditional chutes or "crushes" have been used in Asian range countries for centuries. Figure 5.2 shows a simple but functional chute from an elephant camp in Myanmar that was photographed by the author in 1995.

***Figure 5.2.*** *Restraint chute used in Myanamar. The elephant is walked into the chute and then posts are inserted horizontally behind the elephant to contain it. (Photograph by John Lehnhardt.)*

## TRANSPORTING ELEPHANTS

Transporting elephants should always be done by professionals with well-documented histories of successful elephant moves. There are two options for containment during transport: crating or trailering. In crating, the elephant is loaded into a self-contained elephant crate and then loaded onto a flatbed truck, into a semitrailer, onto an airplane or cargo ship, or into a train car. Trailering requires an elephant to be either restrained in a reinforced semitractor trailer or train car or unrestrained inside a crate/chamber inside a trailer.

The key to successful elephant transport is conditioning the elephant to the transport container and restraints that may be required. The more comfortable the elephant is with the transport container, the less stressful the transport will be. Elephants conditioned to transport, such as performing circus elephants, react to transportation with little or no stress. See Chapter 8 discussion, page 87.

## TRAINING

An effective elephant husbandry program requires that managers have the ability to control the elephant sufficiently to perform appropriate care functions. Training is one of the primary tools for accomplishing these care functions listed in Table 5.1.

### Training Methodologies Used in the North American Facilities

The methods used to train elephants in captivity have become one of the most contentious issues in animal management in North America and around the world. The AZA in the recent past debated whether to mandate a particular elephant management system, generally known as *protected contact,* in its member institutions. AZA did not decide to make such a mandate, but if it had, such a mandate would have essentially banned, in AZA accredited zoos, the traditional method of elephant training and handling, called *free contact*. Free contact is used today for management of almost 16,000 elephants in the care of humans in Asian range countries, an estimated one-third of the world's total Asian elephant population.[4] Also, most elephants in the care of humans in nonrange countries throughout the world are managed through traditional free contact methods. In AZA institutions in North America, however, institutions using protected contact outnumber free contact institutions about 55% to 45%, with the differential increasing about one or two facilities per year.

By definition, protected contact is the training of elephants in which humans and elephants do not share

**Table 5.1.** Care Functions to Be Trained

1. Complete body exam
2. Daily foot inspection and regular care
3. Daily eye examination
4. Daily ear examination
5. Daily open mouth and tongue examination
6. Daily teeth examination
7. Daily tusk examination and the ability to trim tusks without sedation
8. Biological sample collection
   a. Blood
   b. Urine
   c. Feces
   d. Saliva
   e. Skin biopsy
   f. Temporal gland secretion
   g. Trunk wash for tuberculosis culture
9. Accepting injections
10. Accepting oral medications
11. Accepting ear or leg vein catheters
12. Treatment of wounds
13. Enter and stay in the restraint chute
    a. For husbandry procedures
    b. For veterinary procedures
    c. For reproductive procedures
14. Demonstrate a method of restraint if no restraint chute present
    a. For husbandry procedures
    b. For veterinary procedures
    c. For reproductive procedures
15. Accepting an enema
16. Accepting transrectal ultrasound examination
17. Accepting a urogenital examination
18. Accepting semen collection
19. Ability to load and ship for translocation

the same space. Protective barriers and careful positioning are utilized to minimize potential risks to the animal handler from aggression by the elephant. Protected contact uses food as a primary reinforcement to increase the motivation of the elephant to exhibit specific behaviors.

Free contact, by definition, is training of elephants by humans and elephants sharing the same space, without a separating or protective barrier. Traditionally, elephant handlers in free contact use an elephant hook (rather like a stick with a pointed hook on the end) to guide or direct the elephants' movement and to protect themselves from the elephant if necessary. Positive reinforcement with food is also the primary training tool in free contact elephant management. Traditionally, the elephant hook is used as a stimulus tool to elicit behavior and as a negative reinforcer tool to interrupt negative or aggressive behavior.

Opponents to elephant free contact management contend that elephants under this regimen cannot be trained to be in close contact with humans or participate in performances without abusive training methods and learning through fear or the use of force, or even torture. They also contend that managing elephants in free contact is inherently dangerous. By contrast, protected contact is seen by many as nonabusive to the elephants and safer for the elephant handlers.

The question is whether these contentions are true. Is it possible to train elephants without abusive methods, working closely with them, even in the same unprotected space, and have a human-elephant relationship not based on physical punishment, abusive training, and fear of the trainer? Prevalent among opponents of traditional elephant training methods is the assumption that social dominance, established and reinforced through physical means, including inflicting pain or threatening pain, is the only way elephant training can be accomplished. One might ask how it is possible to train elephants at all, and what in their natural history indicates how one might accomplish effective training.

Some definitions are important for this discussion. There is tremendous confusion and misuse of training terms, which leads to misinterpretation of actions and intent.

*Operant conditioning* is defined as learning that occurs when a response to a stimulus is reinforced. Behaviors are conditioned through successive approximation and reinforcement. *Operant* refers to the fact that the subject of the training is the operator, the one doing the behavior, and is actively involved in the training process. *Successive approximation* means building a change in behavior through small increments, which eventually lead to the full behavior.

A *stimulus* is any object or event that causes a subject to respond. An *aversive stimulus* is a stimulus that the subject wants to avoid or escape. Examples of a stimulus may be the presence of a novel item or anything that has allowed the subject to be reinforced in the past. For example, a visual signal (target) or an audible sound (spoken command) is a stimulus. An example of an aversive stimulus may be the touch of an elephant hook or a strong tone of voice.

A *reinforcement* is any action or event that increases the probability that a response will be repeated. Reinforcement of behavior may be positive or negative. The simplest way to understand the difference is to think of *positive* as the addition of something the subject wants and *negative* as the subtraction of something the subject does not want.

Positive reinforcement may be defined as the addition of anything, which occurring in conjunction with a behavior, tends to increase the probability of the behavior occurring again.[20] Examples relevant to elephants would be food (offering a carrot), praise (good girl!), physical affection, or anything the elephant will work to get.

A simple behavior such as lifting the foot could be taught with positive reinforcement in several ways: One method would be by "capturing" the behavior, or reinforcing successive approximations to the desired behavior. The handler can wait until the elephant lifts its foot

even a little and then immediately reinforce it with food, gradually increasing the height required of the foot before reinforcing it until it has reached the desired height for the behavior. A target is a tool the elephant is trained to move toward, usually a stick with a ball on its end. The target can be moved or lifted until the foot rises to touch it. The keeper then immediately reinforces the desired response.

The Elephant Husbandry Resource Guide (EHRG) states, "The handler primarily uses positive reinforcement to teach the elephant each step of the behavior. Positive reinforcement is providing a reward that the elephant desires, such as food and/or praise, when the elephant executes the behavior chosen by the handler. The presentation of the reward must be given to the elephant at the exact moment the elephant performs the behavior in order to communicate to the elephant that the behavior was the one being requested. Timing of the reward is one of the most important aspects of the communication process. Presenting the award too early or too late communicates to the elephant that the behavior it was performing at the moment of the reward was the desired one."[18]

Negative reinforcement is the cessation of an aversive stimulus. After the elephant is given an aversive stimulus, such as the touch of the elephant hook, the hook is removed. To teach the elephant to lift its foot on command, the keeper would touch the back of the elephant's foot with the hook. The elephant doesn't like the touch of the hook and moves its foot away. The keeper immediately removes the hook (negative reinforcement) and may also give the elephant food or praise (positive reinforcement). This can also be done in successive approximations. However, it is the removal of the hook after the elephant has responded that increases the probability that the elephant will repeat the behavior. To reiterate: It is not the aversive stimulus (the touch of the hook) that is the reinforcement, but the removal of the aversive stimulus (the touch of the hook). Other examples of negative reinforcement would be a push from the tusk of another elephant or avoidance of a swat from another elephant's trunk.

"Handlers may also use negative reinforcement to teach an elephant a behavior. Negative reinforcement is often confused with punishment. But the terms do not mean the same thing. Negative reinforcement encourages the repetition of a behavior and punishment is used to extinguish a behavior. Negative reinforcement removes something in conjunction with the performance of the desired action or response. The use of an ankus to cue a behavior can be an example of negative reinforcement. An ankus is a tool used by many elephant handlers and a cue is a stimulus where the response is reinforced. This action can then be rewarded with food and praise using positive reinforcement to further communicate to the elephant that the behavior of moving its leg was correct."[18]

Punishment may be defined as an unpleasant action that occurs immediately after an unwanted behavior. This is done to decrease the likelihood of the behavior recurring. The important concept is that punishment cannot change the behavior it follows, because the behavior has already occurred. It is meant to affect future behavior. Negative reinforcement is meant to have an impact on the immediate behavior and allows for the desired behavior to be reinforced positively. Not providing a food reward when the desired behavior is not achieved is an example of a punishment. The behavior is over, and there is no chance to undo it. However, it is assumed that the punishment influences the subject to display the desired behavior the next time, which then can be reinforced.

Punishment may also be used to establish social or physical dominance. The traditional method of initial training of elephants uses physical punishment first to establish dominance and then shifts to reinforcement training to establish desired behavior patterns.

**Other elephant training methodologies.** There are a few newer and developing elephant training systems that are not widely used but are being experimented with at one or two facilities.

STARS. The Standardized Training and Reinforcement System (STARS), developed by Richard "Army" Maguire, is a free contact training method that incorporates 10 specific movements, called circles and lines, which the handler uses to guide the elephant's movement.[14] This method focuses on the trainer's responsibility for providing appropriate reinforcement to the elephant to receive compliant behavior. The elephant is allowed to make mistakes and given the option to respond appropriately. Primary food reinforcement is used as in protected contact. Though used only in a few institutions, there is one unpublished study that indicates one elephant had reduced cortisol levels after changing from traditional free contact to STARS (personal communication, Mike MacClure, Baltimore, Maryland, January 2005).

Passive control. *Passive control* is a term to describe a nondominant technique that utilizes the barn and yard facilities, keepers' knowledge of the individual elephant, recognition of species-specific behavior and the principals of operant conditioning to encourage an elephant to cooperate. Negative reinforcement, punishment, or tools of control such as an ankus are not used.

Crucial to this system are 1) access to large and diverse spaces, 2) insuring that all basic needs are met, 3) keeper knowledge and attitude, 4) providing elephants with choice, and 5) permitting elephants to respond in their own time frame. In this system the keeper's role is not to dominate or dictate the elephants' lives but to provide a place where the elephants feel safe.[5,6]

Hybrid training methodologies. Increasingly, institutions are mixing protected and free contact methods, both within their programs with different elephants, and even working elephants in both management systems. At one institution (author's personal experience), the elephants have been moved back and forth between protected contact and modified free contact to facilitate management during calving. The expectant mothers are reconditioned to restraints and, when on restraints, worked in modified free contact to assist in the birthing process and introduction of the calf to the mother after birth. This hybrid approach has shown that the elephants can clearly distinguish one method from the other and move easily from one approach to the other with no confusion.

There are many approaches and training methodologies, or tools, used in elephant management. If the goal is to have the best elephant management possible, it is important to use all the tools available.

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# 6 Nutrition

Ellen S. Dierenfeld

## INTRODUCTION

Insight into diet selection, nutrient composition of native feedstuffs, and feeding behavior of free-ranging elephants provides a foundation for husbandry practices in captivity, while contributing to the conservation of the elephant and its natural habitat.

## GENERAL FEEDING ECOLOGY

The natural diet of elephants has been extensively documented, including the African savannah elephant, *Loxodonta africana* africana,[3,4,8,10,22,23,25,26,28,35,36,39,43,60,68,75] the African forest or pygmy elephant, *Loxodonta africana* cyclotis,[8,58,67,77] and the Asian elephant, *Elephas* maximus.[23,47,63,72] These and recent reviews of elephant nutrition[18,49,69] should be consulted for specific details. Despite these numerous studies, however, the proximate factors that influence food selection and foraging patterns in elephants are poorly understood.

Elephants have been described as "generalized feeders" in the wild, consuming more than 400 different species of plants. Other studies, however, suggest they can be extremely selective feeders. Elephants preferentially consume a few botanical taxa, but choices vary widely and are likely highly influenced by region, season, and ecosystem. For example, Viljoen[75] reported that desert-dwelling elephants of Namibia had a preference for woody plant species, irrespective of abundance or plant size. In contrast, Chiaki[10] observed savannah elephants from Tsavo National Park in Kenya choosing grasses, shrubs, and herbs as the main part of their diet. Using spoor analysis, Tchamba and Seme[67] found the diet of forest elephants in Cameroon to consist mainly of grasses and fruits. By direct observation, Sukumar[63] found that just 25 species of plants—ranging from grasses, bamboo, trees such as *Acacia* spp., shrubs, and fruit—constituted 85% of the diet of elephants in southern India.

Grasses have been reported as the most prevalent food chosen by the elephant, with trees, shrubs, leaves, twigs, roots, fruits, seeds, herbaceous plants, soil, and stones comprising varying percentages of their dietary intake.[10,26] During the wet season, elephants tend to be preferential grazers, utilizing sedges and grasses at a time when the protein content of these species is high. However, more browsing occurs in the dry season when the protein content of grass decreases to less than 2.5% and tannins and toxins accumulate.[3,37] A study in Uganda showed grass was present in 99% of 70 elephant stomach content analyses, comprising 25–100% of the stomach content by weight. By comparison, only 10% of the total stomach content of 71 elephants consisted of leaves, twigs, fruit, and shrubs.[8] Herbaceous material and stones comprised 3% of the total stomach material, with a suggestion that stones were apparently being ingested incidentally with plant material and soils.[8,60] High grazing pressure may, however, also be a consequence of habitat conversion from woodland into grassland; dietary habits in both Murchison Falls Park, Uganda, and Amboseli Park, Kenya, changed from browsing to grazing as habitats degraded.[38,44,59]

The influence of season on the extent of grazing was observed in Uganda where grass consumption ranged from 28.6% of the total diet in the dry months to between 57.2% and 97% in the wet months.[8,23] Similarly, for Asian elephants the proportion of time spent grazing ranged from 10%–94%, and browsing time ranged between 6% and 90% in southern India, with variation depending on habitat, time of day, and season.[63]

It has been clearly demonstrated through observational studies,[3,10,25,35,36] spoor analysis,[58,67] fecal analysis,[10,25,77] gastrointestinal studies,[14,25] and isotope ratio studies[9,63,68] that elephants are both grazers and browsers, with fruit and bark contributing a small but significant proportion of the diet. Asian elephants tend to include a higher proportion of grass in the diet compared to African, but the feeding choices of elephants have been attributed to several factors, with variation influenced primarily by habitat and season.

### Feeding Behaviors

Elephants in Tsavo National Park, Kenya, were observed spending 48–63% of daylight hours in feeding activities,[10,22] and Asian elephants observed in Sri Lanka spent 17–19 hours (75–80%) of the day feeding.[72] Elephants in Murchison Falls Park, Uganda, fed continuously throughout the day, stopping only when shade was available for rest.[39] Eltringham[23] observed three feeding peaks in African elephants in a somewhat continuous feeding pattern: the morning, the afternoon, and finally around midnight. Similarly, Vancuylenberg[72] defined three feeding phases, the first described as rapid movement and low feeding, the second as a little movement and high feeding, and finally a certain amount of movement and relaxed feeding. Movement and migration, usually to water and, subsequently, from water in search of food, will reduce the rate of feeding, as will periods spent resting in the shade.[39]

During the time elephants spent feeding on browse, 86% of the activity involved the removal of terminal twigs or leaf stripping, 11% involved breaking the main stems of plants, and 3% of the time was spent debarking, uprooting, or pushing over trees.[58] Similar observations made by Tchamba and Seme[67] found 45% of feeding activity involved grazing, 38% stripping off fruits from trees, 6% the removal of terminal twigs or leaf stripping, 5% debarking of trees, 4% breaking the main stem, and 3% uprooting or pushing over trees. Sukumar[63] noted feeding behaviors varied with the season. For example, when the grass initially appeared, after the first rains, elephants removed the blades in small clumps without uprooting the plant. However, later in the season when the grass attained a height of 0.5–1 meter, entire clumps were uprooted with the trunk and dusted off, the top portion consumed, and the basal portion with roots discarded. Field[25] reported that elephants took considerable trouble in removing soil from the base of grasses, and Buss[8] observed elephants using their front foot to uproot plants and subsequently washing the material by holding it in their trunks and swishing it back and forth in water.

### Geophagy (Eating Soil)

Consumption of soils at specialized licks suggests that licks may be targeted to supplement an inadequate dietary sodium (Na) intake, although concentrations of other minerals, including Ca, Mg, and K, are often higher in lick soils than surrounding areas.[32,54] Recent studies in particularly nutrient-poor soils, the Kalahari-sand region of the Hwange National Park, Zimbabwe, found that elephants did indeed consume high-Na lick soils presumably to balance Na needs, but did not exploit those soils or termitaria for other minerals such as Ca or Mg, which were likely obtained from their green forage. Sodium balance experiments are required to determine Na requirements of elephants, but extrapolations using the horse as a model appeared to fit with the behavioral and intake results obtained. Termite mounds have been shown to be important microhabitats resulting in higher-quality elephant forages—providing better nutrient composition and lower levels of secondary plant compounds—from which individuals may derive over a quarter of their food intake and as such may provide a critical natural resource that warrants further investigation in understanding nutritional balance.[33]

## CHEMICAL COMPOSITION OF NATIVE DIETS

Sukumar[63] found a correlation between seasonal elephant food choices and nutrient composition, specifically the protein and carbohydrate content. Although numerous studies of feeding behavior exist, only a limited number of researchers collected plants for nutrient analysis.[10,25,45,47,60] Others sampled the stomach contents[13,25,43,73] or other gastrointestinal tract sections.[14,73] Blood serum level may also provide some insight into diet quality because it can be an indicator of the nutritional status of animals.[5,6]

### Protein

Reported crude protein (CP) levels in diets consumed by elephants ranged from 2% of dry matter (DM), found in palm leaves from India, to 26–30% DM, found in shrubs from Kenya.[10,47] Seasonal effects on dietary crude protein levels, examined in Uganda, indicate variation from 5.2% DM in the dry season to 12.4% DM in the wet.[43] During the wet season at Tsavo National Park, Kenya, vegetation consumed by elephants contained 8–18% CP for the most part; in the dry season, grasses had 5–7% CP, but legumes and forbs still contained 10–12%.[4] Low urea concentration in elephant plasma correlated with low dietary protein during the dry season.[6] Seasonal protein deficiencies have been suggested, resulting in inhibited growth if the level of digestible protein is below 5–7% of the total diet DM.[43]

Nutrient content also varies by type of plants eaten: Browses in Wankie National Park, Zimbabwe (formerly Rhodesia) ranged from 8–24% CP, with the majority 12–18% (DM basis). Most grasses at the same time contained 3–6% CP, although some had 10–12%.[78] Similarly, leaves of 11 browses eaten during the wet season in Southern India measured 13–26% CP, and grasses in the same season contained 9–10% CP. During the dry season in this report, browse CP dropped to 6–18%, whereas grasses contained about 3% CP.[63]

### Lipid

Field studies have shown that many browse species contain a higher crude fat content compared to captive herbivore diets, and also in comparison to grasses.[21] A diet low in browse, such as the diet of elephants from degraded habitats in Murchison Falls National Park, Uganda, is reported to contain 1.2–1.8% of DM because lipid[43] may be deficient in essential fatty acids.[21,25,43,44]

### Fiber

Crude fiber in leaves consumed by free-ranging African elephants varies between 13–62% dry matter,[22,23,41] with a preponderance of values between 20–50%. Average crude fiber in palm (*Caryota urens*) leaves consumed in India was 24%.[47] The detergent system of analysis, which cannot be directly compared to crude fiber, better characterizes the potential digestibility of fiber fractions by herbivores. The detergent system differentiates neutral detergent fiber (NDF, comprising hemicellulose, cellulose, and lignin), acid detergent fiber (ADF, comprising cellulose and lignin), and lignin—an indigestible fiber fraction.[74] Crude fiber tends to underestimate ADF and overestimate the indigestible fiber fraction (lignin) when both assays are conducted on the same sample. McCullagh.[43] reporting lignin and cellulose values together (equivalent to ADF), showed a variation between 32% DM in the wet season and 41% DM in the dry season. Lignin concentration in plants eaten by elephants in Uganda ranged between 3.1–38.4% DM in the early dry season and 2.6–27.8% in the late dry season, whereas cellulose concentrations showed no seasonal differences, with a range between 21.9–71.8% DM.[10] Fiber concentrations eaten by elephants in South Africa averaged 62% NDF, 48% ADF, and 15% lignin.[45]

### Minerals

Mineral analysis of elephant diets resulted in significant variations recorded both between seasons and among plant species. Calcium, which has received more attention than other minerals, has been found to range from 0.13% DM in the wet season to 0.38% DM in the dry season and from 0.36–1.44% DM in grass-herb vegetation to 0.53–8.92% DM in shrubs.[10,43,60] Bark, with a calcium concentration of up to 5.7% (see also Holdo[32]), has been suggested to serve as a supplementary dietary source of this nutrient.[63] Selected analyses of macromineral concentrations in native elephant foods are found in Table 6.1.

In addition to these data, potassium (K) values ranged from <0.25 to 3.6% of DM in African browses[22,32,34] and 0.4 to 2.4% in three Sri Lankan browses analyzed.[16]

Trace element data have been reported from grasses as well as browse stems and twigs of *Bombax ceiba* and *Ficus religiosa*, eaten by elephants in Nepal.[57] In summary, Cu ranged from 10–39 mg/kg, Fe 152–429 mg/kg, Mn 16–37 mg/kg, Se 0.1–0.4, and Zn 20–52 mg/kg DM in these species.

## DIGESTIVE PHYSIOLOGY

### Intake, Digestion, and Passage

Dry matter intake and digestibility data in 24 published elephant feeding trials on a variety of diets varying in composition (n = 11 for African elephants, n = 12 for Asian elephants, n = 1 for both species fed together) has been summarized.[11] Forty-five individual African elephants, ranging in weight from 504 to 4900 kg, consumed 1.19 to 1.81% of body weight in dry matter daily. Seventy individual Asian elephants (weighing 1555 to 3550 kg) ate 1.03 to 4.4% of body mass, with all but a single animal (fed fresh palm leaves) eating less than 1.65% of body mass in DM daily.

Digestibility in elephants is found to be in the range of 40–60%, with estimates as low as 22–32% for DM in foods consumed by wild African elephants.[29,51,52] Clemens and Maloiy[14] compared apparent digestibility at various sections of the gastrointestinal tract and found the highest DM digestibility value (28.2%) to occur in the upper portion of the colon in his study. The summary feeding trials demonstrate clearly that digestibility coefficients of elephants are lower than those of horses fed diets of similar nutrient composition,[11] due to the significantly faster passage rates of elephants. Dry matter digestion ranged from 22–66% across these trials

**Table 6.1.** Macromineral Concentrations (% of Dry Matter) in Native Plants Eaten by Elephants

| Location | Season | Plant Part | Ca% | P% | Mg% | Na% | Reference |
|---|---|---|---|---|---|---|---|
| Zimbabwe, Hwange National Park | Dry | Mature leaves | 0.02–3.12 | | 0.08–0.64 | 0.002–0.06 | 32 |
| Hwange | Dry | Young leaves | 0.01–1.32 | | 0.10–0.57 | 0.005–0.05 | 32 |
| Hwange | Dry | Stems, twigs | 0.11–1.85 | | 0.02–0.20 | 0.001–0.02 | 32 |
| Hwange | Dry | Bark | 0.13–3.93 | | 0.01–0.33 | <0.001–0.02 | 32 |
| Zimbabwe, Wankie National Park | February | Browse | 0.35–2.47 | 0.11–0.33 | | | 78 |
| Wankie National Park | | Grass | 0.41–0.66 | 0.09–0.20 | | | 78 |
| Malawi, Kasungu National Park | | 12 spp. Tree leaves | | | | 0.10–1.25 | 34 |
| Tsavo National Park | | 59 spp. Browse and grass | 0.37–3.61 | 0.08–0.36 | | 0.01–1.67 | 22 |
| Southern India | Wet | Bark, browse leaves | 0.25–5.72 | | 0.08–0.21 | | 63 |
| Southern India | Dry | Bark, browse leaves | 1.77–3.74 | | 0.07–0.14 | | 63 |
| Southern India | Wet | Grass leaves | 0.19–0.46 | | 0.06–0.08 | | 63 |
| Sri Lanka | Sept–Mar | 3 spp. Browse leaves | 0.07–7.7 | | 0.02–1.0 | 0.02–0.52 | 16 |

(excluding the highly digestible 73% fresh palm leaves); organic matter digestibility ranged from 35–62%. Apparent DM digestibility decreased with increasing fiber content on forage–based hay (both grass and alfalfa) diets.

A rapid gastrointestinal (GI) transit time has been recorded in elephants, ranging from 21–55 hours in captive Asian and 21–46 hours in captive African elephants fed forage-based diets.[11,29,39,51] Interspecifically, mean retention times correlate positively and significantly with body size in captive elephants; thus larger elephants also achieve higher fiber digestibility. However, there appears to be a possible interspecific difference in passage rate. Asian elephants retain food longer in the digestive tract relative to body size and achieve higher digestibility coefficients compared to African elephants—possibly due to ecological adaptations for divergent dietary strategies. Nonetheless, low apparent diet digestibility overall and high fiber profiles of many of the plant species eaten by elephants underlie the large amounts of vegetation needed by the elephant to meet its nutritional requirements. Not only must quantity, but also quality, of the diet be considered.

Comparison of fermentation products (VFAs, volatile fatty acids) in Asian elephants on hay-only diets suggests that elephants are adapted to diets with higher fermentation potential than the grass hays used in the captive study.[11] The VFA ratios of free-ranging elephants are not indicative of "concentrate"-type feedstuffs but rather achieve their higher concentrations through greater (or faster) fermentation of a different fiber substrate than forages commonly fed in captivity. Browse may indeed be a critical substrate for meeting the maximum energy potential through fermentation in elephants, given the rapid passage characteristics measured. This may be particularly relevant to consider in a free-range or high-energy environment.

## Mineral Nutrition

Mineral nutrition of elephants remains an area requiring further study. The limited published analyses on mineral content of browses, grasses, and bark eaten by wild elephants (Table 6.1 and above) suggest that horses may provide a good physiologic model for elephants in terms of mineral requirements, and elephants should be able to meet these estimated requirements through selective feeding behaviors in various habitats.

Regarding captive feeding, reports of mineral deficiency diseases including rickets in a hand-reared elephant[24] and possible zinc deficiency associated with foot lesions[56] appear rather isolated in the literature. Calcium requirements of 8–9 g/day have been determined for tusk growth in male elephants, and up to 60 g of calcium daily has been estimated as necessary for a lactating cow to meet the growth needs of her calf.[43,63]

Calcium metabolism in elephants appears similar to that of horses, with absorption averaging approximately 60% independent of dietary concentration in both equids and Asian elephants.[11] Excess Ca is excreted through the urine in horses, and high urinary Ca concentrations have been reported in captive elephants.[53] Absorption of Ca is significantly decreased by the addition of pelleted feeds in both equids and elephants.[11]

## Vitamin Nutrition

**Fat soluble vitamins.** The conversion of ß-carotene to retinol for vitamin A activity has not been investigated in detail in the elephant, and circulating ß-carotene concentrations have been found to be undetectable in all elephant plasma or serum samples examined (Dierenfeld unpublished).[57,61] Vitamin D status has likewise not been examined in detail.

Vitamin E deficiency has, however, been reported in captive elephants with low tissue levels of this nutrient,[19] leading to various studies of absorption and effectiveness of different supplements.[50,65,76] Positive and rapid responses to micellized[76] and water-soluble[50] vitamin E preparations have been documented in elephants, although more recent investigations suggest that the isomeric form of vitamin E may be as, or even more, important to absorption than the carrier matrix per se. Using isotopically labeled natural or synthetic vitamin E administered simultaneously, Swanson[65] found that bioavailability of natural vitamin E is about twofold greater than synthetic in both African and Asian elephants. Additionally, a species difference in metabolism was apparent, with African elephants showing both a more rapid uptake of natural vitamin E and a slower rate of decline of both forms compared with Asians.

True metabolic disparities may underlie any differences in expected circulating levels of both α-tocopherol (as a measure of vitamin E activity) and retinol (vitamin A activity) between elephant species (see Table 6.2), but have not been clearly demonstrated independent of dietary influences in controlled trials.

Nonetheless, normal circulating concentrations of these fat-soluble nutrients remain relatively low in both free-ranging and zoo elephants compared with domestic livestock models, including equids, with European values in the same ranges as from animals in U.S. zoos.[20,27,55] Circulating concentrations of retinol cannot be used as an assessment of vitamin A status in elephants or other species, because blood values correlate poorly with storage of this nutrient in critical tissues. Although serum or plasma tocopherol concentrations are somewhat better correlated with vitamin E status, the inherent biological variability and low overall concentrations suggest that results of a single assay should be interpreted with caution in any case.

**Water soluble vitamins.** Very little data is available on water-soluble vitamin nutrition in elephants. Improved horn and nail strength in elephants given daily oral doses of 75 mg biotin per kg body weight is reported

**Table 6.2.** Circulating Vitamin A and E Concentrations in Elephants

| Species | N | Vitamin A (Retinol; µg/l) | Vitamin E (α-Tocopherol, µg/ml) | Reference |
|---|---|---|---|---|
| **Zoo** | | | | |
| African (North American) | 223 | 80.0 ± 60.0 | 0.47 ± 0.75 | 20 |
| African (European) | 57 | 57.5 ± 15.3 | 0.33 ± 0.46 | 27 |
| Asian (North American) | 945 | 60.0 ± 40.0 | 0.50 ± 0.36 | 20 |
| Asian (European) | 20 | 50.7 ± 7.1 (n = 22) | 0.24 ± 0.16 | 27 |
| **Free-Ranging** | | | | |
| African | 38 | 50.0 ± 30.0 | 0.50 ± 0.25 | 20 |
| African | 70 | 39.0 ± 7.0 | 0.61 ± 0.27 | 55 |
| Asian | 12 | 50.0 ± 10.0 | 0.27 ± 0.0 | 20 |
| Asian | 26 | 63.0 ± 3.0 | 0.77 ± 0.05 | 57 |

anecdotally[27] but has not been experimentally demonstrated. Single oral doses of biotin administered to Asiatic elephants followed the same pharmacokinetic curves as horses and were likely poorly absorbed and rapidly excreted.[71] Proper GI function, fermentation, and microbial synthesis would be expected to provide natural sources of this B vitamin, without a need for external supplementation. Reported improvements in foot health associated with biotin supplementation are frequently confounded by alterations in foot husbandry; documentation of direct effects of nutritional supplementation requires further, properly designed, investigation.[70]

## NUTRIENT REQUIREMENTS AND RECOMMENDATIONS FOR FEEDING

Taking into account the unique physiologies that have been identified in elephants, and until controlled experiments are conducted to determine actual requirements, minimum nutritional requirements are still based primarily on extrapolation from published dietary guidelines for the domestic horse (Table 6.3). Until further information is forthcoming, the National Research Council[48] recommendations for diet formulation should be considered the basis of ration formulation for elephants at differing physiological stages. Essentially no comparative information exists for elephants at differing life stages, with the exception that the digestible protein requirement for growing (10 years old, 1337 kg) elephants was calculated from balance trials at 1300 g/day, compared to an adult (37 yr, 3605 kg) requirement of 2370 g/day.[1]

Detailed practical feeding guidelines (amounts, concentrate product formulation specifications, and suitable hay composition data) are detailed elsewhere[49,69] and should be consulted. Good quality hay should form the basis of the feeding program, with browse, produce, and supplements comprising the remainder. Even the growth requirement of 1300 g protein per day can be met on a diet containing 8% crude protein, with a DM digestibility of 50%, at intake levels of 1.3% of body weight. Dietary protein levels below these guidelines, or poor hay quality, may lead to protein deficiency, illness, and death.[70]

Results of a diet survey in U.S. zoos[2] involving 17 institutions and 56 elephants (27 Asian, 29 African) found that an average of 50 kg DM was consumed daily, primarily grass hay (timothy). Other grasses (orchard, brome,

**Table 6.3.** Modified Minimum Nutrient Concentrations (DM Basis) in Elephant Diets, Based Largely on Extrapolation from Nutrient Requirements of Horses[48] (from Olson 2004)[49]

| Nutrient | Maintenance, Breeding, Early Pregnancy | Late Pregnancy, Lactation | Growth |
|---|---|---|---|
| Crude protein, % | 8–10[a] | 12–14[b] | 12–14[c] |
| Lysine, % | 0.3 | 0.4 | 0.4–0.5 |
| Calcium, % | 0.3 | 0.5 | 0.5–0.7 |
| Phosphorus, % | 0.2 | 0.3 | 0.3–0.4 |
| Magnesium, % | 0.1 | 0.1 | 0.1 |
| Potassium, % | 0.4 | 0.4 | 0.5 |
| Sodium, % | 0.1 | 0.1 | 0.1 |
| Sulfur, % | 0.15 | 0.15 | 0.15 |
| Copper, mg/kg | 10 | 10 | 10 |
| Iron, mg/kg | 50 | 50 | 50 |
| Manganese, mg/kg | 40 | 40 | 40 |
| Selenium, mg/kg | 0.1 | 0.2 | 0.2 |
| Zinc, mg/kg | 40 | 40 | 40 |
| Cobalt, mg/kg | 0.1 | 0.1 | 0.1 |
| Iodine, mg/kg | 0.6 | 0.6 | 0.6 |
| Vitamin A, IU/kg | 3000 | 3000 | 3000 |
| Vitamin D, IU/kg | 800 | 800 | 800 |
| Vitamin E, IU/kg | 100–150 | 100–150 | 100–150 |
| Thiamin, mg/kg | 3 | 3 | 3 |
| Riboflavin, mg/kg | 3 | 3 | 3 |

[a]Adult maintenance, 8% CP; breeding bull, pregnant cow (first two trimesters), 10% CP.

[b]First year of lactation, 14% CP; 2nd year of lactation, 12% CP.

[c]Weanling, 14% CP; 3-year-old, 13% CP; 4-year requirements of horses[48]; from old[49] to 12-year requirements of horses[48]; from old[49], 12% CP.

Sudan, Bermuda, prairie, meadow) were also used, depending on geographic locale. Hay alone comprised approximately 40–80% of dry matter intake, with the average nutrient composition 7% crude protein, 68% NDF, and 41% ADF. Only 4 of the U.S. institutions mentioned browse as a regular component of the feeding program; one facility reported daily intake of 43% (as-fed basis) browse, another fed 44% produce (as fed weight).

Concentrate pellets were used to balance vitamins and minerals in the diets, but cannot replace forage as the primary fiber source essential for proper GI function and fermentation. Nine of the surveyed institutions fed a general herbivore pellet, whereas seven used a concentrated elephant supplement. No follow-up comparative health or reproductive studies on these survey groups have been reported. Pellets were offered at about 10% of the total diet by weight. Produce offerings also averaged approximately 10% of the diet by weight, ranging in general from 3–21%. A number of supplements were mentioned regularly, including salt (both loose and in block form), Zn, vitamin E, biotin, and generic vitamin/minerals or trace mineral blocks. When fed a properly formulated diet, based on forage and concentrate pellets of known composition, the need for additional supplement products should be carefully evaluated.

Calculated nutrient need for mature maintenance (Table 6.3) could be met primarily through good-quality forage; added protein and nutrients required for growth or lactation could be met through replacement of a portion of the grass hay with higher-quality alfalfa if necessary, but many institutions report no change in the basal diet throughout pregnancy and lactation, with no apparent problem. When hay mixtures are not adequate to meet the energy, protein, mineral, and vitamin needs, consider adding a formulated pellet in appropriate quantities to balance out the diet. Hence knowledge of forage quality and consistency must be the primary focus of an elephant feeding program (as outlined specifically in Olson[49] and Ullrey[69]).

A survey of British and Irish zoo elephant diets (2001 unpublished) that encompassed 15 institutions and 34 animals reported that grass hay formed the staple forage (primarily meadow, or timothy), supplemented with commercial equid diets, vitamins (primarily vitamin E), and fruits and vegetables. No pregnant, lactating, or growing animals were included in the survey, and diets did not distinguish between Asian and African elephants. Nonetheless, results did differ considerably from those reported from the U.S. Zoo Survey[2] in several ways: Only one institution reported feeding ad libitum, the average meal offerings were three times daily (range one to seven). Dry matter intake ranged from 0.7–2.9% of body mass, with 25–100 kg DM consumed daily. Calculated nutrient composition of the U.K. diet was 14% CP, 60% NDF, and 34% ADF—higher protein and lower fiber compared to the U.S. diets. Notably, 12 of 15 facilities reported feeding browse regularly, although no proportions were given. Sodium, Zn, and vitamin E were determined to be low.

A further survey of diets fed in European facilities (personal communication, Nijboer) documented 13 institutions in the Netherlands. In general, these zoos offered more supplements, concentrates, produce, and browse to elephants compared to U.S. zoos. Fiber level (reported as crude fiber) was lower than in North America, but because a different assay was used, direct comparison is difficult. A high incidence of diarrhea and obesity has been documented as problematic in European zoo elephants, attributed to excessive use of concentrates in the diet.[30]

A list of browses fed to elephants in the U.S. is found in Olson,[49] but nutrient analyses have not been conducted on all species. Browses may assist in meeting mineral, vitamin, and other needs in elephants, and also provide enrichment. However, its full impact on animal health, reproduction, and behavior in captive feeding programs has not been systematically evaluated and remains a topic for further investigation.[62]

## POTENTIAL LINKS BETWEEN NUTRITION AND REPRODUCTION IN ELEPHANTS

### Body Condition

Obesity is a problem in captive zoo elephants, linked to the consumption of overly digestible diets and reduced physical activity. This high prevalance of obesity in western zoo elephants may be related to poor reproductive output and performance[66] affecting both males and females. Overcondition is also believed to be a contributing factor in long labors, dystocias, stillbirths, and ultimate deaths of both calf and cow in many instances.[49] Elephants in good physical condition and exercised frequently are reported to have shorter labors and easier deliveries. Under natural environmental conditions in Sri Lanka, relative body weight (kg/cm at shoulder) in elephants was positively correlated with reproductive output.[17] Adaptation and adherence to standardized body condition indices are suggested for improved captive management of elephants.[31,49]

### Iodine Status

Iodine metabolism of elephants needs to be investigated in more detail, with both essential free-ranging and captive components. It is suggested that the reproductive potential of a given elephant population depends notably on the availability of this element, and that the supply or restriction of iodine-rich water bore holes offers a feasible and humane method of controlling animal numbers in a given habitat in situ.[46] An as-yet untested hypothesis remains that elephants (and other large herbivores) utilize mineral licks in response to iodine content. The exceptionally large brain and body size of elephants may result in a special sensitivity to subclinical iodine deficiency and possibly an increased

iodine requirement in this species that may be met only through management intervention. Captive studies may be used to investigate the iodine requirement of both Asian and African elephants, examine urinary and fecal excretion of iodine, and establish indicators of physiologic status that may then be field tested and validated in elephant populations of long-term known demographics.

### Dietary Fatty Acids and Sperm Membrane Stability

When wild animals are brought into captivity, a nutritionally based shift in fatty acid composition is seen. Overall, polyunsaturated fatty acids (PUFAs) are reduced and the proportion of n-6 fatty acids increases, while the proportion of n-3 fatty acids decreases.[12,15] Dietary deficiencies in linoleic acid and linolenic acid are associated with impaired spermatozoa development,[40,42] and differences in the levels of 22:6, n-3 have been associated with a differential response of elephant spermatozoa to cryogenic freezing.[64] At this time, spermatozoa collected from Asian elephants cannot be cryogenically preserved, and spermatozoa collected from African elephants can be cryopreserved.[7] Investigations into fatty acid metabolism and dietary supplementation trials are needed with both Asian and African elephants to determine whether such supplements might 1) influence the levels of membrane PUFAs within elephant erythrocytes and spermatozoa, 2) have an impact on spermatozoa fertility parameters (motility, motility after cryogenic freezing/thawing, acrosome integrity), or 3) alter lipid metabolites (serum triglycerides and cholesterol) and/or the antioxidant status of serum and sperm.

## RESOURCES

The following references contain useful detailed discussions of many of the topics mentioned in this chapter: Clauss,[11] Dierenfield,[21] Olson,[49] and Ullrey[69].

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# 7 Preventive Health Care and Physical Examination

Susan K. Mikota

## INTRODUCTION

Health issues are a major concern to the survival of elephants. In range countries where captive and free-ranging elephants are in close proximity or commingle, the threat of disease transfer is enhanced. Habitat stress has been correlated with pathology in wild African elephants,[35,36,37] and captive elephants are confronted with numerous health issues.[26] The risk of disease transfer in cooperative breeding programs has raised growing concern among zoological facilities striving to develop self-sustaining captive populations.[31] This chapter discusses preventive health care measures for captive and free-ranging elephants and physical examination procedures.

## PREVENTIVE HEALTH CARE

Health is a state of physical and psychological well being. Numerous biological, physiological, and environmental variables determine whether health or disease will prevail. Proper nutrition and housing, an appropriate social environment, sound disease control measures, and other positive husbandry practices support health. Poor hygiene or diet, a lack of exercise (or overwork), and stress predispose to disease. The characteristics of a potential pathogen (virulence, infecting dose) and the status of the individual animal (age, sex, immune competence) are additional determinants.

Wild animals often do not manifest clinical signs until disease is well advanced. The masking of clinical signs, though serving to protect weak animals from predation, complicates the diagnostic and therapeutic efforts of zoo and wildlife veterinarians. Thus measures to prevent diseases are paramount to successful elephant care. Recommended components of a preventive health care program for captive elephants are described in Table 7.1. Additional guidelines are included in Appendices 12, "Guidelines for a Comprehensive Elephant Health Monitoring Program (AZA/SSP)" (which includes guidelines for herpesvirus testing), 13, "Quarantine Guidelines for Elephants (AZA/SSP)," and 14 "Recommended Elephant Preshipment Guidelines (AZA/SSP)." See Chapter 11 for a list of infectious diseases of elephants.

## EVALUATION OF THE ELEPHANT PATIENT

Elephants may live 50–70 years,[42] and most will experience health problems at some point in their lives. Determining a definitive diagnosis can be a challenging task for the veterinarian confronted with a sick elephant. Although some limitations exist, the approach to patient evaluation is the same for elephants as for other large mammals. The basic steps are 1) record a thorough history, 2) identify the presenting complaint, 3) conduct a physical examination, 4) establish a prioritized list of differential diagnoses, and 5) conduct appropriate diagnostic tests and assess results to establish a tentative diagnosis and therapeutic plan.

A basic knowledge of the age, gender, and geographic or environmental variables associated with specific problems may aid diagnosis. Degenerative arthritis, for example, is common in older zoo elephants. Floppy Trunk Syndrome is seen primarily in free-ranging African male elephants. To date, elephant pox virus has been diagnosed in Europe but not in North America. Anthrax, although reported in captive elephants, is much more likely to occur in free-ranging animals. Angry villagers may poison crop-raiding elephants, but toxicities are relatively rare in captive situations.

### Normal Versus Abnormal

Veterinarians that provide medical care for elephants should have a basic understanding of elephant biology and be able to distinguish normal from abnormal signs. Anatomy and physiology unique to the elephant is described in the chapters corresponding to each body system. The basic vital signs are listed in Table 7.2. (a more comprehensive list of physiological parameters is pre-

**Table 7.1.** Recommended Components of a Preventive Health Program for Captive Elephants

| | |
|---|---|
| Quarantine | Quarantine should be in accordance with guidelines established by the relevant zoo or government agency.[a] |
| Preshipment screening | Preshipment screening for parasites and infectious diseases may prevent disease transmission between elephants and facilities. Preshipment screening is particularly important for situations where quarantine may not be possible.[a] |
| Physical examination | Conduct physical examination of captive elephants at least annually; include weighing and blood collection for hematology, serum chemistries, serology, and banking. Periodic reproductive evaluation and foot radiographs are advised.[a] |
| Fecal examination for parasites | At least annually, more frequently if parasitic disease is a problem. |
| Routine deworming | Advisable if parasites are a recurrent problem; frequency determined by extent of problem. Most parasitologists advise alternating anthelmintics. |
| Annual tuberculosis screening | Culture of respiratory secretions by the trunk wash procedure is the recommended TB screening technique used in the United States as of 2005. The procedure is described in Chapter 13. Refer to the current Guidelines for the Control of Tuberculosis. Serological testing may soon be available. The intradermal tuberculin test is not accurate in elephants. |
| Foot care | Routine foot trimming is necessary in most captive situations. Frequency will vary with the enclosure substrate, exercise, and individual animal. Although trimming is usually carried out by elephant caretakers, veterinarians should examine the feet at least quarterly. Baseline radiographs are recommended. |
| Diet evaluation | Review diets at least annually and as part of the workup for medical problems, particularly those presenting with GI signs (refer to Chapter 6). |
| Behavioral enrichment | Elephants are highly intelligent, and behavior enrichment is important to their overall health and well being. See Chapter 4. |
| Vaccination | Vaccination will depend on exposure risks. Tetanus, anthrax, and rabies are known pathogens for elephants for which vaccines are available; however, published data on protective doses and titers are sparse.<br>Tetanus: In a preliminary study, measurable titers against tetanus were achieved in Asian elephants vaccinated with a 1 cc dose of equine tetanus toxoid followed by a booster at 4 weeks. The titers remained elevated for >1 year; however, the appropriate vaccination interval has not yet been determined. Annual vaccination is commonly practiced, although it is likely that the duration of immunity may be longer.[b]<br>Rabies: Measurable titers (up to 1:1100 in RFFIT)[c] against rabies have been achieved in African elephants vaccinated with a single 2 cc dose of killed rabies vaccine IM (IMRAB 3, Merial, Duluth, Georgia, USA, www.us.merial.com). Titers persisted at >1:50 for at least 2 years. Based on this preliminary data, a dosage regimen of 2 cc killed rabies vaccine IM every 2 years is recommended.[d]<br>Encephalomyocarditis virus (EMCV): Protective titers against EMCV have been achieved in African elephants using an experimental vaccine.[19,18]<br>Anthrax: Prophylactic vaccination against anthrax is recommended in India using an anthrax spore vaccine administered subcutaneously in the caudal fold. The recommended dose varies with age as follows: elephants 2–5 years (1 ml); 5–10 years (1.5 ml); 10–15 years (2 ml); 15–20 years (2.5 ml); 20 years and older (3 ml).[7] |
| Rodent control | EMCV is transmitted by rodents and can be acutely fatal to elephants. Rodent control is currently the most effective means to prevent this disease. This disease has been reported in the southern U.S. and in free-ranging elephants in South Africa, but it has the potential to appear in other areas. Consider vaccination in problem areas. |
| Sanitation and disinfection | Providing a clean, dry area is essential for proper foot health and disease prevention. Frequency of cleaning will vary with each facility, but elephant areas should be cleaned once daily at a minimum. Foot baths are recommended to minimize transmission by fomites. |
| Staff health monitoring | Annual tuberculin test; banking of serum for retrospective studies if possible. |

[a]See Appendixes for guidelines developed by the Species Survival Plan of the American Zoo and Aquarium Association.

[b]Personal communication, Dr. William Lindsay, Orangeville, Ontario, April 2005.

[c]The Rapid Fluorescent Focus Inhibition Test (RFFIT) is a laboratory test that detects rabies virus neutralizing antibodies. The threshold for a protective titer in animals has not been established; however, a titer greater than 1:5 is considered protective in humans. This is the test used at Kansas State University; it is not a species-specific assay.

[d]Personal communication, Michele Miller, DVM, PhD, Orlando Florida, April 2005.

sented in Appendix 5, "Elephant Vital Signs and Physiological Parameters"). The veterinarian should be familiar with signs of health and disease in elephants. These have been described[7,15] and are summarized in Tables 7.3 and 7.4.

## History

A thorough history should be obtained before undertaking a physical examination. Information regarding prior problems or events may be as pertinent to a diagnosis as the presenting complaint. Inquiries regarding diet, housing, exercise, and foot care should be routine and will help assess the quality of care. Record pertinent information concerning previous medical problems. Ask about behavior, attitude, appetite, feces, and urine. Establish the duration and progression of signs related to the presenting complaint. Have any medications already been given? What were the results? Has there been

**Table 7.2.** Elephant Vital Signs

| | |
|---|---|
| Temperature (rectal)[5,6,20] | 36–37°C (97–99°F) |
| Heart rate (beats per minute)[20] | 25–30 (standing)<br>72–98 (lateral recumbency) |
| Respiratory rate (breaths per minute)[33] | 4–12 |

**Table 7.3.** Signs of a Healthy Elephant

| |
|---|
| Constant motion—ears flapping, tail or trunk swinging and swaying |
| Eyes clear and bright; a small amount of clear discharge from the conjunctival sac is normal |
| Mouth, tongue, and inside of the trunk a rosy pink |
| Tip of the trunk moist |
| Skin soft and resilient |
| Moisture present at the base of the nail |
| Neither too fat nor too lean |
| Appetite good, appears content |
| Well-formed dung, brown in color (color may vary with diet); a normal amount is passed with no evidence of straining |
| Urine copious in amount, faintly yellow, with a pleasant odor; no straining during urination |

**Table 7.4.** Signs of an Unhealthy Elephant

| |
|---|
| Listless, decreased movement, unusual behavior, exercise intolerance |
| Dull or sunken eyes, increased tear flow, thick discharge |
| Mucous membranes pale, muddy, bright red, or dry |
| Discharge from the trunk, coughing, abnormal respiratory sounds |
| Dry skin, loss of elasticity, wounds |
| Weight loss, sunken abdomen, prominent ribs (see body condition index) |
| Deceased appetite, anorexia |
| Change in urine or feces (amount, color); straining |
| Lameness |
| Obvious pain |
| Any unusual swelling or protrusion |

a change in routine or environment? Determine the presence of any environmental or social stressors that may be manifesting as physical signs. Provide the owner or handler with the opportunity to contribute additional observations—a dedicated elephant caretaker is often an astute observer.

## Physical Examination

Before starting a physical examination, the handler should be queried regarding the elephant's temperament. In the case of a bull, the perineum should be observed for obvious swelling and the temporal gland openings for swelling and/or discharges to determine whether the bull is in musth. A bull in musth should never be approached by a stranger.[32] The veterinarian should verbally communicate to the elephant before actually touching him, the voice exuding confidence and compassion. The body language of the elephant and particularly the appearance of the eyes may reveal the elephant's mood to an experienced veterinarian (personal communication, Dr. K. K. Sarma, Assam, India, March 2005). Some elephant handlers in the U.S. prefer that the veterinarian not speak directly to the elephant.

**Table 7.5.** Checklist for Physical Examination

| Subjective | Objective | Body Systems |
|---|---|---|
| General condition | Temperature | Cardiovascular |
| Demeanor | Pulse | Endocrine |
| Appetite | Respiration | Gastrointestinal |
| Skin | Weight | Integumentary |
| Head | Feces | Musculoskeletal |
| Eyes | Urine | Nervous |
| Ears | Capillary refill time | Reproductive |
| Mouth | Auscultation | Respiratory |
| Teeth | Rectal (if indicated) | Special senses |
| Mucous membranes | | Urogenital |
| Trunk | | |
| Tusks/tushes | | |
| Forelimbs | | |
| Feet and nails | | |
| Thorax | | |
| Abdomen | | |
| Hindlimbs | | |
| Genitalia | | |

The physical examination should be conducted in a consistent manner, using the same method each time. All body systems should be included. It is important to perform a thorough examination, even with the early discovery of an obvious abnormality, because many problems are multisystemic. A physical examination checklist is presented in Table 7.5. If the tuberculosis (TB) status is unknown or TB or other potentially zoonotic diseases are suspected, the examiner should consider wearing protective clothing and using protective equipment, including gloves and a hepa-filter mask.

One approach to an examination, preferred by the author, is first to observe the elephant from a distance and then conduct a close exam starting at the left side of the head. The exam proceeds along the left side of the body to the rear of the elephant and then to the right side, ending at the head. All body areas are observed or palpated. A detailed description follows.

Begin by observing the general body condition and attitude of the elephant from a distance of 2–3 meters (6.5–10.0 feet). Walk completely around the elephant to observe from multiple directions. The elephant should be neither too thin nor too heavy. Temporal depression, protrusion of ribs or scapular spines, and a sunken flank area may typify poor condition. A body score index for Asian elephants is presented in Table 7.6. Is the elephant alert and active? Is there evidence of pain or swelling? Record any identifying scars, characteristics, or structural abnormalities. Note the condition of the skin and any wounds throughout the procedure. The normal

**Table 7.6.** Asian Elephant Body Condition Index[a]

| Body Area | Observation | | | Score |
|---|---|---|---|---|
| 1. Head: temporal depression (view from several angles) | Full and convex in outline when viewed from behind, frontal ridge vaguely outlined = 2 points | Slightly to moderately concave, frontal ridge defined = 1 point | Deeply concave, frontal ridge forms a craterlike rim around the temporal depression = 0 points | |
| 2. Scapula (shoulder blade) (view from side) | Spinous process not visible or slightly visible = 2 points | Spinous process visible as a vertical ridge with a concavity between the ridge and the posterior edge of the scapula = 1 point | Spinous process pronounced and bladeline with the acromial process appearing as a knot = 0 points | |
| 3. Thoracic region (view from side) | Ribs not visible, barrel smooth = 2 points | Some ribs visible, but the extent and demarcation not pronounced = 1 point | Many ribs strongly demarcated with pronounced intercostal depressions = 0 points | |
| 4. Flank area (immediately in front of pelvis) (view from side and behind) | | No depression visible, flank bulges outward in front of the pelvis = 1 point | Depression visible as a sunken area immediately in front of the pelvis = 0 points | |
| 5. Lumbar vertebrae (behind ribs and in front of pelvis) (view from behind; an elevated vantage point may be necessary) | Not visible, lower back smooth and rounded = 2 points | Visible as a ridge; skin slopes away from the top of the ridge; height of the vertebrae does not exceed width = 1 point | Visible as a knifelike blade; sides of the spinal ridge are parallel, and the height exceeds the width = 0 points | |
| 6. Pelvic bone (external angle of the ilium) and rump (view from several angles) | Not visible (or slightly visible); rump region between ilium and caudal vertebrae filled with tissue (and not forming a depressed zone) = 2 points | Visible but not pronounced; the rump is slightly depressed between the ilium and the caudal vertebrae = 1 point | Visible as a jutting bone; rump is a pronounced sunken zone between the ilium and the caudal vertebrae = 0 points | |
| 7. Axillary fat (immediately behind joint of humerus and scapula) | The SQ contains a thick handful of fat, easily seized = 2 points | The SQ contains some fat = 1 point | The skin thin and little tissue palpable beneath = 0 points | |
| 8. Brisket fat (between forelegs at base of neck) | Sternum well padded with muscle and fat; bone neither visible nor palpable = 2 points | Sternum not visible but palpable = 1 point | Sternum both visible and palpable = 0 points | |
| 9. Tail | | Fat and muscular, not bony feeling = 1 point | Thin and bony, feels stringy, individual joints palpable = 0 points | |
| Total | | | | |

0–5 = emaciated condition
6–10 = average condition
11+ = very good condition (or fat)

[a]Developed by Dr. V. Krishnamurthy, Dr. C. Wemmer, and John Lehnhardt. Adapted from personal communication, Dr. V. Krishnamurthy, India, 2000. A version of this table appears in Das, ed. 2003. Healthcare, Breeding and Management of Asian Elephants. New Delhi, Project Elephant. Govt. of India, p. 189.

healthy skin is soft and wrinkled, uniformly warm to touch, free from scurf, almost black in color, and it has no appearance of glaze along the side of the spine or the hip.[7,13] The skin over the nails should be moist from sweat. This can be tested by applying dust, which should stick (personal communication, Dr. K. K. Sarma, Assam, India, April 2005). Return to the left side to begin the close examination. A flashlight can be useful if the inspection is being performed inside a barn. Inspect the head and trunk. Wounds on the forehead may be evidence of improper handling. Fibrosarcomas, although not common, may occur on the trunk and may appear as multiple growths (see Chapter 18, Fig. 18.9). Observe the eyes for abnormal ocular discharges, corneal scars, or cataracts and the conjunctival color for anemia, icterus, or cyanosis. The pupils are normally constricted, making it difficult to check a pupillary light reflex unless the elephant is moved to a darkened area. The lachrymal gland is replaced by the Harderian gland. The lachrymal duct is vestigial, and therefore tears naturally spill over onto the face from the corner of the eye.[38] Many elephants resist close physical examination of their eyes.

The tusks/tushes should be examined closely—asymmetry of growth, discoloration, or foul smell should arouse suspicion. If there is death of pulp/nerve owing to injury, the affected tusk will stop growing. Injury to the tusk may also cause discoloration, and a

fissure may act as a portal of entry of infection into the pulp, causing pulp decay. This may lead to inappetance and loss of condition. Lifting the flap of skin fold overlying the tusk allows a view of the base of the tusk (this may be a site for parasite eggs, e.g., Cobboldia, seen in Asia).

Note the two small temporal gland openings on either side of the face caudal to the eye and above the zygomatic arch. The temporal gland will enlarge and drain during musth in bulls. Drainage from the temporal gland is also noted in cows that are excited or in estrus. The temporal gland may be a site of infection (see Chapter 18, Fig. 18.1).

Listen for any audible respiratory sounds. There is an area at the base of the trunk that inflates and deflates. This is the location where the elongated nostrils enter the skull and air is directed into the trachea. Note any discharges from the trunk. If the elephant permits its trunk to be handled, check for symmetry of airflow between the two nares. Many elephants will raise their trunk on the request of their handler, permitting examination of the oral cavity. Observe the tongue and mucous membranes, which should be pink. Check the capillary refill time (1–2 seconds is normal) by applying light digital pressure to the mucous membranes. Exercise caution when placing a hand into the mouth as elephants can clamp down and inflict serious injury.

Examine the teeth. The dental formula is I 1/0, C 0/0, PM 3/3, M 3/3 (26 teeth total throughout the life of the elephant). The 24 molars (6 in each arcade) erupt in the caudal jaw and progress rostrally. Generally only 2 teeth are in wear at any one time. The emerging molar pushes the older molar forward and segments (laminae) break off. Loose sections of tooth may be observed during this process. Although age may be determined by dentition,[14,23,24,30,38] it is not easy to do so antemortem. Aging is discussed in the digestive chapter.

As the examination proceeds to the side, palpate the auricular artery on the caudal aspect of the ear to evaluate and count the pulse. The normal heart rate in a standing elephant is 25–30 beats per minute. Lateral recumbency greatly increases the rate. Record the pulse rate. Observe the ears for scars. Tears or holes in the exterior pinnae may be identifying features and should be recorded. Lice, if present, are commonly observed behind the ear. The ability of the elephant to close the ear canals precludes the usefulness of ear thermometers.

Observe the forelimbs for wounds, asymmetry, swelling, heat, or tenderness. The orientation of the bones of the limb is almost vertical, leaving little definition on the exterior to separate the arm, forearm, or manus. Pay particular attention to the foot because problems in this area are among the most common ailments of captive elephants. A thick keratinized pad covers the sole (slipper) on the palmar and plantar surfaces. The rounded forefoot is semidigitigrade. There are five digits on both front and back feet; however, the number of toenails differs. In general, Asian elephants have five toenails on the front feet and four on the rear. African elephants have four toenails on the front feet and three on the rear. Individual variation does occur, and the number of toenails should be recorded. Check for toenail cracks or overgrowth of the nail or cuticle. Brush or wash the foot to permit a more thorough examination.

Examine the skin of the foot for lacerations, contusions, or abrasions that may result from injuries or improper chaining. Additional conditions affecting the foot include foreign bodies, contusion of the nail, nail bed infection, and abrasion of the sole. Snare injuries may be seen in free-ranging elephants. Pododermatitis (infection of the foot) is particularly serious for elephants because it may progress to osteomyelitis that is often refractory to treatment.[16] Note that the sole has ridges and grooves providing a distinctive footprint. These ridges are normal and may prevent injury from small pebbles. Bruises or puncture may not be visible unless the sole is clean.

Proceed to the side. Auscultation of the heart should be performed at this time. The heart is more cranial compared to other ungulates and auscultation of heart activity is not easily accomplished in an adult elephant. If the elephant is trained, ask the handler to move the left forelimb forward or better still, have the elephant lift the left forelimb. Using an electronic stethoscope (Littmann® electronic stethoscope model 4000, 3M Health Care, St. Paul, MN 55144, 1-800-228-3957, www.3M.com/Littmann), which amplifies sound up to 18 times, may ameliorate these limitations. Note the character, depth, and frequency of respiration by observing movements of the chest. The normal constant motion of the elephant complicates this task, and respiratory sounds cannot easily be heard by auscultation. If a pulsoximeter is available, oxygen saturation can be measured (normal is 96.2 ± 1.55%[18]), although this is not a necessary component of a basic exam. Observe the mammary glands in the pectoral area on the ventral thorax just medial to the elbow. Note the abdominal outline to detect any distention or ventral edema.

Evaluate the hindlimb as for the forelimb. The hindfeet are an elongated oval and semiplantigrade. Thoroughly inspect the hindfeet.

Observe the genitalia for any evidence of discharge. Bulls in musth typically dribble urine; the preputial rim may be pale or light green or there may be a greenish preputial discharge. The vulva is located ventral to the pelvic symphysis, rather than near the anal opening as in other mammals.

Elephants urinate 5–10 times/24 hours[4,39] and the 24-hour urine volume may be 25–53 liters.[4,8,11,39] The urine may appear clear, straw-colored, or cloudy.

The temperature may be taken rectally or by inserting a thermometer deep into a freshly voided dung bolus. The average temperature is 97–99°F (36–37°C).[5,6,20] The temperature of a fresh fecal bolus gives a measure of

body temperature accurate to 0.5°.[6] Temperatures in excess of 100°F (37.8°C) should be considered elevated.

Asian elephants defecate 12–20 times/24 hours and typically pass 5–8 boli/defecation.[8] African elephants may produce as much as 100 kg of dung over a 24-hour period.[9] Feces should be well formed but moist. Hard feces may indicate dehydration or impaction, and the absence of feces may indicate a blockage. Mixing a small amount of feces with water may be used to detect excessive sand, a common cause of impaction or colic. Rectal examination is not part of the routine physical examination but is indicated if there is a history of gastrointestinal problems. Use well-lubricated shoulder-length gloves.

Proceed to examine the thorax and abdomen on the right side of the elephant and continue toward the head on the right side. Finally observe the elephant during movement. Lameness or pain may be detected only with motion. If equipment is available, weigh the elephant. Platform scales suitable for elephants are available commercially and some units are portable. Truck scales may often be borrowed from local highway departments. Weights range from about 2000–5500 kg (4400–12,100 lb) for Asian elephants and from 2000–7000 kg (4400–15,400 lb) for African elephants.[34] Body weight may be estimated and a number of formulas have been developed for Asian[2,8,22,40,41] and African[12,21,24] elephants. No formula is sufficiently accurate for all age groups and the degree to which the weight of an individual elephant may be over- or underestimated can be significant.[27] Obtaining weights using scales is preferred whenever possible.

In the author's experience, the following formula by Hile[17] shows reasonable correlation with scale weights in adult Asian elephants:

$$\text{Wt (kg)} = 18.0(\text{HG}) - 3{,}336$$

where *HG* (heart girth) is the circumference of the chest just caudal to the front legs. The average error for adult elephants is 4–5%. The average error increases to ± 15% in elephants <13 years old, and the formula is not recommended for very young elephants.[17] Other formulas for estimating weight are discussed in Chapter 13.

Methods for taking standard measurements have been described for Asian elephants and should be included when physical examination is conducted for scientific purposes.[43]

## HEALTH MONITORING IN FREE-RANGING POPULATIONS

Health monitoring in free-ranging populations requires a methodical approach with different criteria than are used in disease diagnosis in an individual elephant. The routine collection of basic epidemiological data (morbidity, mortality, onset of disease outbreaks, etc.) must be correlated with causative etiologies that may include infectious agents and environmental, ecological, physiological, and pathological factors. Body condition indices may also help to monitor noninfectious conditions such as starvation and malnutrition (see Table 7.6). In African elephants, the depth of the lumbar depression and protrusion of the adjoining dorsolateral ridge of the wing of the ilium may be used to assess body condition, whereas the buccal depression and temporal dent are more indicative of age.[1]

Infectious diseases require additional evaluation using microbiological, pathological, serological, and parasitological techniques.[25] A combination of invasive and noninvasive and pre- and postmortem methods may be required. Molecular analysis of stress-activated proteins is under investigation and may prove a useful tool to detect environmental stress and compromised health.[3]

Surveillance of commingling or adjacent wild or domestic animal populations is important because diseases may cross over from these populations. In the event of an infectious disease outbreak, vaccination at the disease interface may be required. In India, the Wild Life Protection act of 1972 dictates the vaccination of domestic livestock within a 5 km radius of protected areas to prevent infectious diseases such as anthrax, foot and mouth disease, and hemorrhagic septicemia from spreading to wildlife.[29]

It is also important to establish the health status and disease prevalence of source and release populations prior to translocation operations. Detailed guidelines have been developed for African elephants.[10] Data collection may include physical examination; biometric measurements; samples for parasitology and genetic studies; and blood for hematology, serum biochemistry, and serology. Consideration should be given to banking blood and tissue samples for later study.

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# 8 Physical Restraint and Handling

Murray E. Fowler

## INTRODUCTION

It is incumbent upon a person who takes the responsibility (moral and legal) of manipulating an elephant's life to be concerned for its feelings, the infliction of pain, and psychological upsets that may occur from such manipulation. One must be objective about such manipulations, but be certain that it is in the best interest of the elephant.[9,27] Every restraint procedure should be preceded by an evaluation as to whether the procedure will result in the greatest good for that elephant. The following questions should be answered:

1. Is it safe for the handlers?
2. Is it safe for the elephant?
3. Will the procedure accomplish the objective?
4. Will it return the elephant to prerestraint status or improve it?

Restraint and handling procedures may constitute some of the most stressful episodes of an elephant's life if done incorrectly. Handlers and veterinarians should understand the physiologic basis of stress (see Chapter 17 for more details on stress).

A veterinarian should never attempt to examine or perform any procedure on an elephant without the assistance of a qualified handler. The veterinarian must assess and have confidence in the handler's ability to deal with the elephant. Lacking that confidence, a veterinarian should discuss it with a person in charge or postpone the procedure.

Asian elephants have been in domesticity for over 4,000 years. If they have been properly trained to work with people they are generally safe, gentle animals.[20,37] However, they are large and may be unmanageable in certain situations. Regardless of the elephant management method used, any human/elephant contact is inherently dangerous.[5] All available precautions suitable for an institution or facility should be evaluated and utilized whenever possible. Staff should be properly trained and experienced in the professionally recognized forms of elephant management and should be constantly aware of the risk of human injury and death.

The care of captive elephants has improved exponentially during the last two decades as more elephants are trained to accept veterinary procedures. Handlers are becoming more knowledgeable about elephant behavior and training. Zoo administrators now realize that resources must be allocated to provide time for handlers to train their charges.[43]

Thirty years ago, if the feet of a zoo elephant needed to be examined or trimmed, the elephant may have been tethered to a fence and the foot physically lifted and anchored in an elevated position. Now the elephant places its foot voluntarily on a pedestal, tub, spindle or bar at a protective barrier to allow inspection and/or manipulation.

## OFFENSE AND DEFENSE

No one except a trained, qualified elephant handler should approach, come in contact with, or command an elephant. Elephants will not listen to or follow the commands of a stranger. Elephants use several methods for offense and defense, including biting; slapping with the trunk; and grasping with the trunk and pulling, pushing, or throwing. Elephants may purposely step on a person's foot, and they are adept at kicking and can easily balance on one front and one hind leg.[34] Extreme aggression may be exhibited by the elephant kneeling and head-pressing upon what they perceive as a threat, inconvenience, or toy. Even an elephant in an elephant restraint device or on tethers may injure a person unfamiliar with an elephant's reach or its signals of aggressive intent.

Although the swinging tail is usually not considered an offensive weapon, it must be considered when administering medication in the rear quarters or when tethering a hind leg. Being soundly struck hurts and a blow to the face or head could be injurious.

The foregoing is said not to frighten veterinarians or handlers, but rather to impress upon them the fact that these normally gentle giants are so strong and so large that serious injury or death may result from improper assessment of an elephant's behavior.

## BEHAVIOR

All personnel working with elephants should understand basic elephant behaviors. The reader is referred to Chapter 4 for a detailed discussion of behavior. Pay particular attention to the ears and trunk to assess the mood of an elephant. Following are behaviors to be aware of:[25]

**Alert.** The elephant stands facing a person with the head raised, ears spread, tail raised, trunk raised, or trunk turned in a "sniff" position.
**Kick.** An elephant may strike forward with a forelimb or toward the side or rearward with a hindlimb.
**Mock charge.** The elephant runs toward another elephant or a person with ears extended, head and tusks held high, and the trunk extended; tail may or may not be elevated. The charging elephant stops before reaching the target and usually trumpets.
**Real charge**. The trunk is tucked under the head, the head is up, and the elephant attempts to contact the target with the head. The ears are usually close to the head and usually there is no trumpeting.
**Slap.** An elephant strikes another elephant with the trunk.
**Sniff.** The trunk is extended down and forward in a "J" shape, with the tip out horizontally to sniff another elephant or person (Fig. 8.1).
**Wariness.** The elephant is in heightened alertness, and with eyes wide open it glances at other elephants.

All these behaviors are noteworthy because people may be severely injured if these behaviors are directed toward them.

### Vocalization

Over 30 vocalizations have been distinguished in African elephants, but only 10 have been studied in Asian elephants. Vocalizations to be aware of during restraint procedures include the following:

**Bellow**. A loud fear or pain-related call.
**Blow**. An audible air blast from the trunk or a visual blast containing dust or food particles.
**Scream**. Produced when an elephant is extremely excited or angry.
**Trumpet**. Loud, high-frequency, pulsating sound.
**Musth rumble**. A deep-throated, guttural, or bubbly vocalization that is loud and low.

Musth is a normal periodic behavior in mature male elephants[6] (see Chapter 32). For safety, handlers must recognize the primary signs of musth, including aggressive behavior, drainage from the temporal glands, dribbling urine from the prepuce, and unusual vocalization (musth rumble). Other signs that are not unique to musth, but commonly occur during musth, are anorexia, dehydration, and somnolence. A bull elephant in musth is dangerous and should be handled only from behind a protective barrier.

***Figure 8.1.*** *An elephant sniffing to gather information.*

## TRAINING

Modern elephant management programs emphasize training based on positive reinforcement that makes the elephant a willing participant in the handling procedures.

This book is not meant to be a definitive treatise on training of elephants. (For more details, see the following references in the References section at the end of this chapter: 1,2,17,25,30,32,43.) Training is an integral part of successful handling of elephants. Elephants gather information and respond positively or negatively, which is called *learning*. If a person initiates and coordinates the process, it is called *training*. Elephants are intelligent animals, capable of learning many procedures if trainers are consistent.

Training an elephant is challenging because it relies completely on effective communication between the elephant and the trainer, using the language of actions and consequences. It is highly recommended that one handler only be given the responsibility of training a

new behavior. Using more than one handler to train may introduce inconsistencies in the training process, which may cause confusion and anxiety on the part of the elephant. Each elephant facility should have a written elephant training protocol under the direct supervision of the elephant manager.

Handling elephants directly (*free contact*) or through a barrier (*protected contact*) requires different training regimens, but the end result is to be able to carry out a procedure in a safe and efficient manner for both the elephant and humans. Some institutions use a hybrid of these two management strategies.

Having a well-trained elephant requires that the trainer also be trained and experienced.[28,30] The person must be dedicated to improving the well being of his or her charges and be willing to learn from others and attend training seminars and workshops. Elephant caretakers should be permanently assigned to work with elephants so that mutual respect and trust may be fostered.

A trained elephant is likely to be safe to work around with a minimum of physical or chemical restraint being required. Training the elephant to position its body, ears, limbs, tail, or head to allow examination, collection of laboratory samples, or administration of medication greatly enhances the care that may be provided.

The key to an optimal training program is to facilitate opportunities for the elephant to make associations through consequences that enhance understanding of the handler's requests.[25]

*Operant conditioning* is a learning method in which a particular response is elicited by a stimulus because that response produced desirable consequences (*reward*). As an example, if a desired behavior is followed by something the elephant seeks (praise, food, treat) it is more likely to repeat that behavior and even to enhance the behavior. This is called *positive reinforcement*. The presentation of the reinforcement must be given to the elephant at the exact moment the elephant performs the behavior in order to communicate to the elephant that the behavior was the one being requested.[25] Timing is important. Using the foregoing logic, a trainer may move an elephant into an elephant restraint device (ERD) by slowly but consistently asking the elephant to move forward and reinforcing (rewarding) the behavior. See Table 8.1 for some selected terms used in training.

Reinforcers may be positive or negative. Positive reinforcement increases response probability by the presentation of a positive stimulus following a response. *Negative reinforcement* does the same in reverse, through the removal, reduction, avoidance, or prevention of an aversive stimulus following a response.

Verbal commands may be accompanied by touching a specific area of the body (Fig. 8.2, Table 8.2). Trainers working with an elephant behind a barrier usually use a whistle or a clicker as a *bridge* for reinforcement. The bridge is used as a signal to the elephant that the requested behavior has been performed and the reward is forthcoming when the reward cannot be presented immediately.[25]

Elephants may also learn by observing other elephants carry out the desired behavior. It is interesting to watch young circus elephants mimic their elder companions during play time.

It may be helpful if a veterinarian is familiar with some of the commands used by trainers to induce an elephant to perform various behaviors that have relevance to restraint and/or physical examination.

By knowing commands and the appropriate response of the elephant, a veterinarian may be able to evaluate the competence of the trainer/handler. However, the veterinarian should not make the mistake of trying to issue commands. If the elephant is not re-

**Table 8.1.** Selected Training Terms

| Term | Definition |
|---|---|
| Conditioned response | A type of learned response that occurs through association with a specific stimulus. |
| Cue | A stimulus that precedes a behavior, signaling that a specific response will be reinforced if performed correctly. The result is that the stimulus will consistently elicit only that particular response. |
| Negative reinforcement | A process in which a response increases in frequency due to the avoidance, escape, or removal of an aversive stimulus from the animal's environment. |
| Operant conditioning | A type of learning in which behavior is determined by its consequences; it is strengthened if followed by reinforcement (positive or negative) and diminished if followed by punishment. The animal's behavior is instrumental in acquiring the desired response. |
| Positive reinforcement | The process of following an action or response with something that the elephant wants, thereby causing an increase in the frequency of occurrence of that behavior. |
| Punishment | An act that occurs immediately after a behavior it is meant to affect and causes a decrease in the frequency of that behavior. |
| Reinforcer | Anything that occurs immediately following a behavior that tends to increase the likelihood that the behavior will occur again. |
| Stimulus | Anything that elicits or affects a behavioral response. |
| Time-out | Cessation of all reinforcement immediately following an inappropriate or undesirable response. A gentle type of punishment of short duration. |
| Unconditioned stimulus | A stimulus that elicits a particular response without any prior association. That is, it is not a learned association; it is a reflex. |

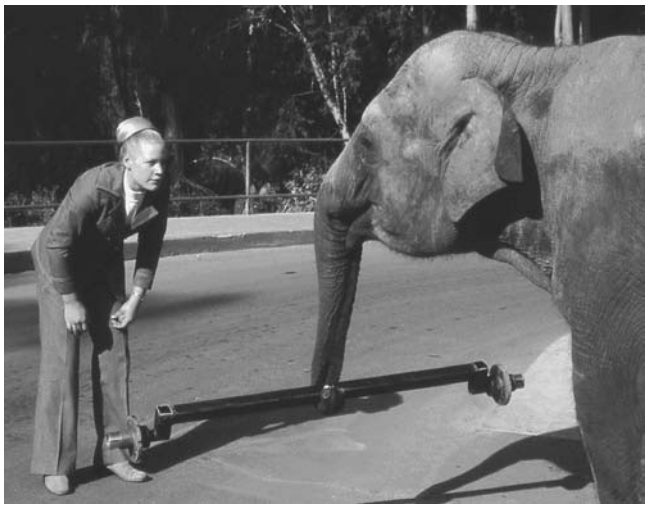

***Figure 8.2.*** *An elephant responding to verbal commands.*

sponding to the handler's commands, it is unsafe for interaction and the veterinarian should leave the elephant area immediately.

## PHYSICAL RESTRAINT WORKING DIRECTLY WITH THE ELEPHANT (FREE CONTACT)[7,8,9]

Elephants should respond to voice (Fig. 8.2), visual and pressure commands, and cues. Minimal restraint may be applied to a calf or juvenile by grasping the ear. An elephant may be trained to place a foot on a spindle or tub (Fig. 8.3), lift a foot (Fig. 8.4), open the mouth (Fig. 8.5), or lie down (Fig. 8.6).

Elephants can rest standing up, but most will sleep soundly for a short time to several hours each day by lying on a side with their trunk coiled. Some elephants may never be observed lying down; in fact they may not be able to lie down or get up because of musculoskeletal problems. One female didn't lie down for 2 years as a result of poly-osteoarthritis. A female in a large zoo supposedly failed to lie down for 10 years because of arthritis in an elbow. It is important to make certain if an elephant is to be laid down that it is capable of rising. A conscious elephant may stay in a sternal position (stretched) for some time; however, if the elephant is chemically immobilized it must be pulled into lateral recumbency within 20 minutes to prevent respiratory embarrassment.

When an unanesthetized elephant is in lateral recumbency, avoid standing or kneeling between the fore and rear legs. If an elephant tries to stand up it will quickly swing the rear limb forward and backward with great force and may injure a person. A keeper at a zoo suffered fractured ribs from being in an inappropriate position.

**Table 8.2.** Commands Used in Elephant Management* (Olson 2004)

| Command | Meaning |
|---|---|
| All right | Release from previous command. |
| Back up | Move backward in a straight line. |
| Come here | Move to the handler. |
| Come in | Move laterally toward the handler. |
| Ear | Present ear forward or through an ear hole in a barrier. |
| Foot | Front leg, foot to elbow parallel to the ground; rear leg, foot to stifle parallel to ground; present foot for chaining; move foot into the foot hole in protected contact. |
| Get over | Move laterally away from the handler. |
| Give | Hand object to the handler. |
| Lean in | Position body parallel to and in contact with a barrier in protected contact. |
| Leave it | Drop whatever is in the trunk. |
| Lie down | Assume lateral recumbency. |
| Move up | Move forward in a straight line. |
| No (quit) | Stop unwanted behavior. |
| Open | Open the mouth wide for visual inspection. |
| Salute | Raise trunk and foot simultaneously. |
| Steady | Freeze. |
| Stretch | Assume sternal recumbency. |
| Target | Move toward target, touched with target pole. |
| Trunk down | Drop trunk straight down to the ground. |
| Trunk up | Curl trunk up to touch the forehead. |
| Turn | Pivot in a circle, right or left. |

*These verbal commands may be accompanied by touching an appropriate spot on the head, body, or legs.

### Use of the Guide

The *elephant guide* has been used for centuries to communicate with elephants and assist in their management. The guide was previously called a *bull hook,* or *ankus,*[1,9,32] but these terms don't denote the proper use of this tool and should be discarded. Examples of similar types of tools include the lead shank on a horse, use of a cane to direct a pig in a show ring, a collar and lead on a dog, leg pressure in dressage horse riding, a child on a leash in a crowd to prevent the child from becoming separated from a parent, or a parent directing a child by a hand on the shoulder.

The guide is a tool used to apply pressure to stimulate a specific response (lift a foot, move right or left, lift the trunk, lie down). Touching with a guide is a cue (signal) to begin a maneuver. Cues are used in the training of all kinds of animals.

The guide is made of a metal rod (preferably stainless steel) or cut from a metal sheet approximately 1 cm (3/8 in) thick. The straight prong has a side arm that makes it possible either to give a push or pull cue. The prongs are tapered to a narrow point, but should not be so sharp as to penetrate or lacerate the skin easily. The metal tip is attached to a wooden, fiberglass, nylon, or metal handle 30–90 cm (12–36 in) long.

The elephant guide should not be used indiscriminately because this is inhumane and exemplifies poor

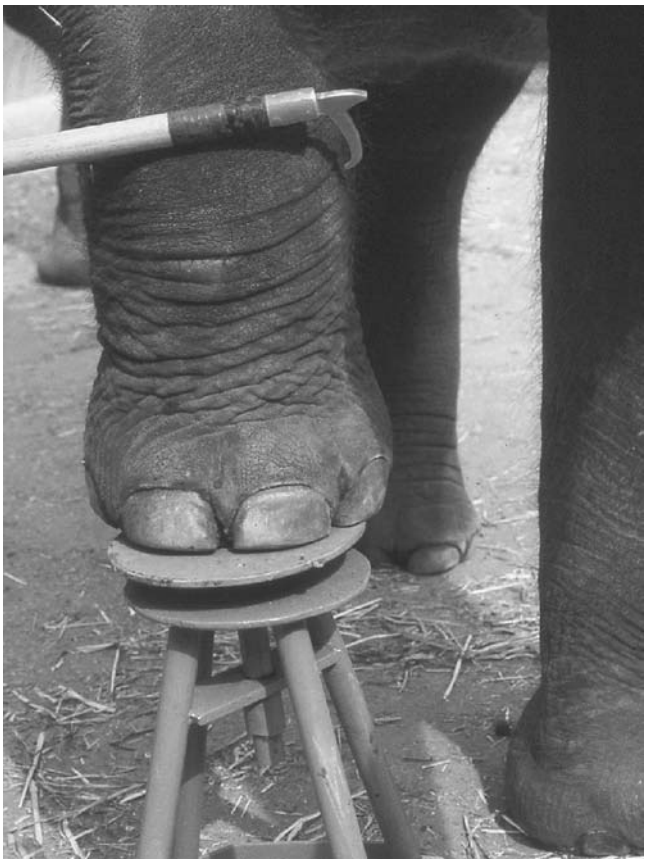

***Figure 8.3.*** *A foot placed voluntarily on a pedestal.*

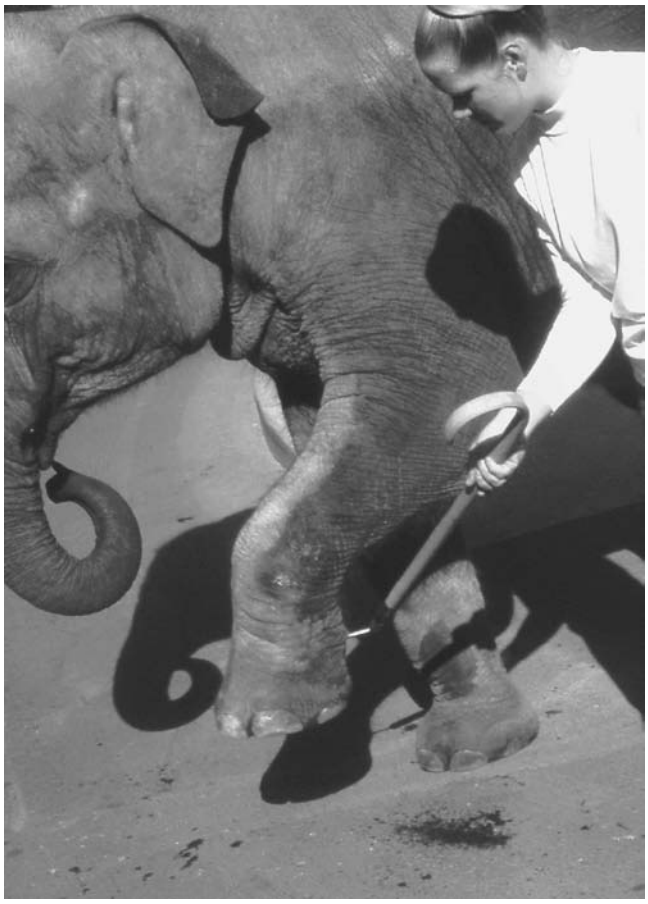

***Figure 8.4.*** *An elephant lifting a foot on cue from a guide.*

training techniques. The ultimate goal of training is that the elephant will respond to a verbal command alone so the guide is rarely necessary.

## Leg Restraint and Tethering[4]

All elephants maintained in captivity should be trained to accept tethering on all four legs, whether or not the elephant is routinely tethered.[3,9,12] A tether is a tool used to restrict the movement of the elephant for specific reasons. The tethering procedure should begin in infancy so that the elephant becomes accustomed to standing quietly while tethered. Chain is the common material used for elephant tethers. Rope may be used in place of chain when training a calf. A soft double-braided or cotton rope, 3.2–3.8 cm (1.25–1.5 in) in diameter, avoids abrasions and contusions. Ropes should be kept clean and dry. Fabric softener may be used to keep the ropes soft and pliable. A sharp knife should be available at all times when using rope for tethers to free the elephant quickly in case of an emergency.

Handlers using rope should be practiced in using knots, hitches, and splices so that in a pressure situation ropes can be secured and released quickly and expertly. Examples of chain usage with animals include tethers for long-term control of dogs and chain shanks on lead ropes for controlling high-spirited horses.

A tether is a tool used to confine an elephant, serving the same purpose as a horse in a box stall, a pet dog or cat confined to a cage or room of a home, or a dog tethered to a dog house. Tethering keeps the elephant from wandering, provides security for other animals and people, provides order and routine for elephants, provides a special space for the elephant, and enables conducting routine procedures, such as toe nail trimming, administration of medication, and cleaning the area.

Chain is used instead of rope or cable because a chain is less easily tangled, is stronger and thus better for use with elephants, doesn't collect moisture (urine) as a rope or even a cable would do, and is more flexible. The desirable diameter of the metal rod used to construct the chain for elephant confinement is 5/16–1/2 inch. The chain links are welded (see Table 8.3). Chains used to restrict movement in a male in musth should be heavier. Chains are only as strong as their weakest links, which are usually the swivels, snap hooks, Brummel hooks, and slot links, so the connections should be checked daily.

Routine tethering has one front leg and the opposite hindleg tethered. Front leg tethers are attached to the lower leg between the foot and the carpus (wrist). Frequently an anklet or bracelet is placed on the leg and

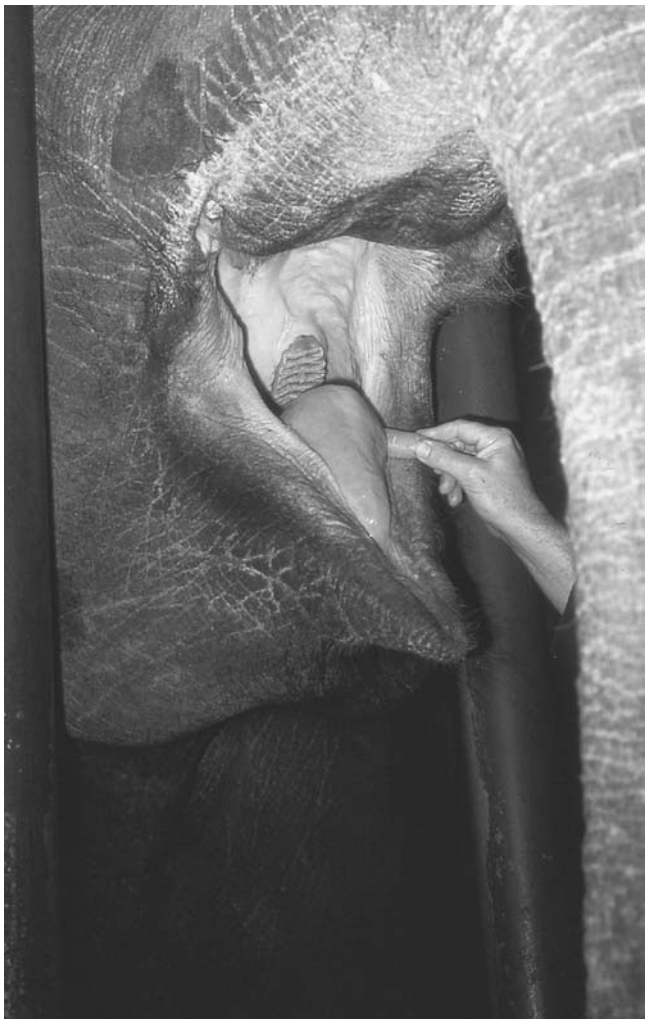

***Figure 8.5.*** *An elephant opening its mouth on command.*

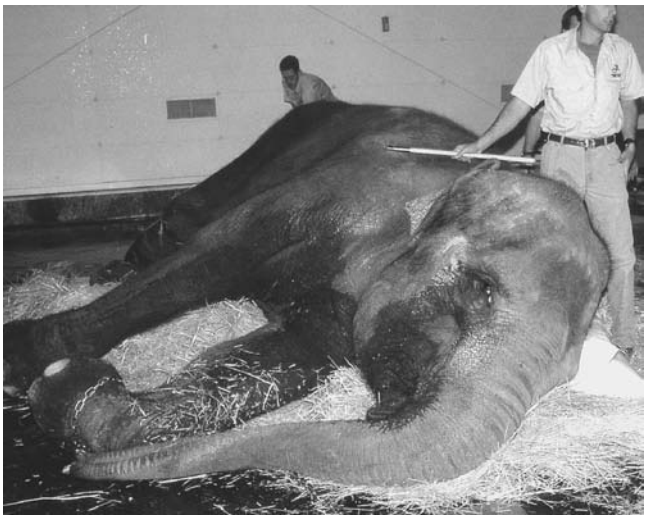

***Figure 8.6.*** *An elephant lying down on command.*

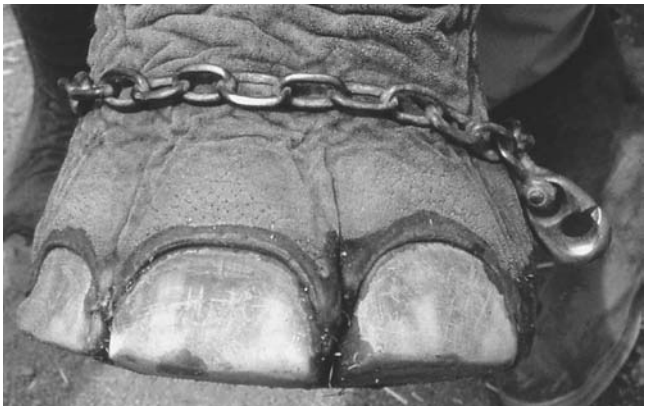

***Figure 8.7.*** *A Brummel hook attached to a bracelet.*

the tether is attached to the anklet (Fig. 8.7). The different shape of the hindfoot allows a tether to slip off the foot; therefore, the hindleg tether is attached above the hock (tarsus) and around the tibia (Fig. 8.8). Alternate legs should be tethered routinely. Tethers should be long enough to allow the elephant to lie down and rise to its feet easily, but not so long as to allow the elephant to turn around and become entangled. Swivels should minimally be used on front leg tethers to prevent the tether from binding as the elephant moves. Bolt cutters should be readily available to free the elephant quickly in case of an emergency.

The use of tethers for confinement of elephants is often maligned by those who do not understand the training and management of animals (companion animals, livestock, horses, elephants). Unfortunately,

**Table 8.3.** Specification on Selected Chain

| Product | P 8 Alloy Steel, Heat Treated, Highest Quality | P 43 High-Test, Low-Carbon Steel | P 70 Transport, High-Strength, Heat-Treated Steel |
|---|---|---|---|
| 5/16″ working load, kg/lbs | | 1769/3,900 | 2132/4,700 |
| 3/8″ working load, kg/lbs | 3220/7,100 | 2449/5,400 | 2994/6,600 |
| 1/2″ working load, kg/lbs | 5443/12,000 | 4173/9,200 | 5125/11,300 |

Source of chain: Peerless Chain Co., 416 E. Sanborn St., P.O. Box 5349, Winona MN 55987-5349. Phone: 507-457-9100. FAX: 507-457-9187. Email: custserv@peerlesschain.com.

***Figure 8.8.*** *A tether attached to the hindlimb above the hock.*

tethers have a negative connotation because of the public perception of chains being used for enslavement. Pictures of chain gangs of prisoners working on roads or railroads come to mind, or of dangerous prisoners entering a courtroom in chains.[22,23] Some states have legislated the length of time that an elephant may remain tethered. It is desirable to have the elephant free of tethers as much as possible. Many facilities no longer tether, but should maintain the elephant training and human skills to do so when necessary. Tethering should not be something that is attempted in an emergency situation.

Each elephant facility should develop a routine tethering procedure and both experienced and new handlers should be required to follow the procedure so the elephant becomes accustomed to the routine. Tethers should be checked daily for signs of wear and worn links replaced immediately. It is unwise to tether some elephants and allow others to mingle freely with them because tethered animals are at a disadvantage in aggressive episodes.

Tethering is neither inherently evil nor necessarily injurious to elephants. A tether may chafe the skin in the area of the leg encircled, but this may be prevented by enclosing the anklet (bracelet) with a segment of discarded canvas fire hose.

Numerous methods are used for attaching tethers to the leg. A clevis may be used to attach the bracelet to the tether, or the end of the tether may be wrapped around the leg and clevised to a link of the chain. These methods are slow and should be used on only well-trained elephants, or the person should be behind a protective barrier while the clevis is attached. Some elephants become quite adept at unscrewing the bolt from a clevis.

Faster methods incorporate the use of special hooks, such as the Brummel hook which may be obtained from marine sailboat outfitters. A permanent anklet is attached to the elephant's front leg with a Brummel hook as part of the anklet. Another Brummel hook is attached to the end of the tether. When the elephant is in the proper position the two hooks are joined rapidly because the hooks have no moving parts (Fig. 8.9). Stainless steel Brummel hooks are preferable to bronze due to strength and rigidity. Bronze is weaker and softer. Another type of hook is illustrated in Fig. 8.10.

## Swaying

When standing and not otherwise engaged in some activity, both captive and free-ranging Asian and African elephants rock back and forth.[9] This is called *swaying* or *weaving,* and some people equate this with undesirable stereotypic behavior.[15,19,33,34] Although exaggerated swaying may be stereotypic, it is also a normal behavior, and in the process of swaying back and forth the elephant is facilitating the circulation of blood from the distal extremities to the heart. In the wild, this function is accomplished during the day as elephants walk many kilometers in a day. Blood flows peripherally easily, but return flow must overcome the gravitational stagnation of blood in the long limbs. This is aided by compressing the digital cushion in the foot, which acts as a peripheral pump to force blood around in the foot and up the

***Figure 8.9.*** *Sequence showing the interlocking of Brummel hooks.*

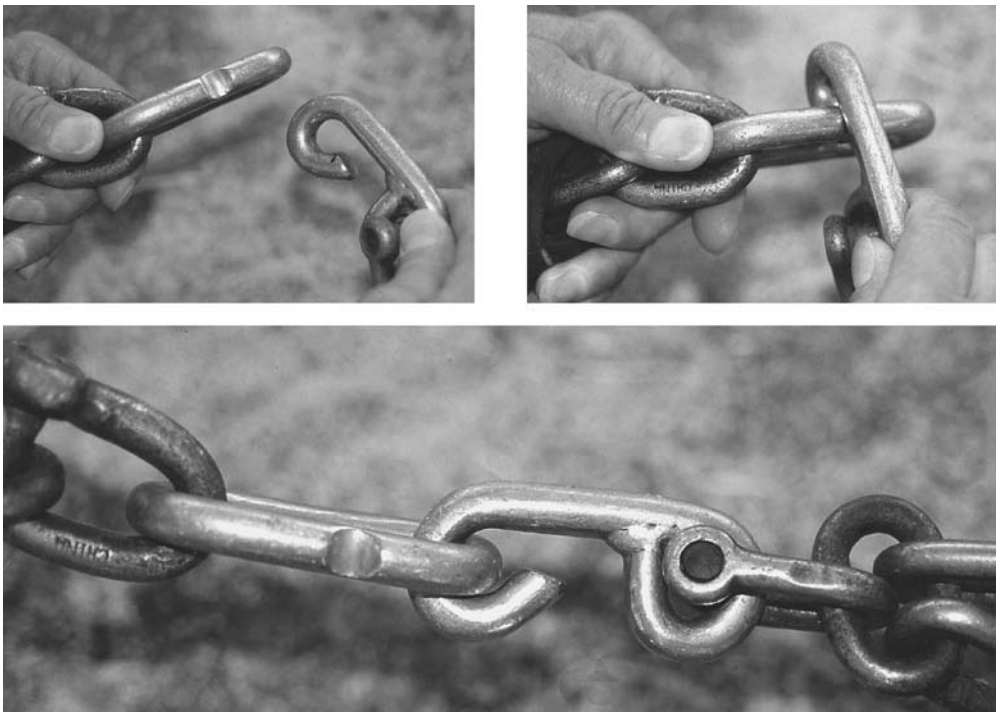

***Figure 8.10.*** *Sequence showing interlocking of another type of hook.*

leg. As the elephant alternates its weight, it is facilitating circulation in the feet and legs.

In a small study conducted on several elephants, as weight was applied, the circumference of the foot just above the nails increased from 5.0 to 11.4 cm or by 7.0 to 9.7%.[11] As weight is applied, the digital cushion compresses and pushes peripherally, causing the increase in circumference and at the same time compressing the veins in the foot, forcing blood up the leg.

## PHYSICAL RESTRAINT BEHIND A BARRIER (PROTECTED CONTACT)

Many North American zoos have begun managing their elephants from behind a barrier,[24] wherein the elephant and the handler do not occupy the same space and the elephant is free to move away from an activity at any time. Training involves giving rewards for appropriate behavior, which may involve placing a foot through a portal for inspection or nail trimming or presenting some other part of the body. Handling in this method is not entirely injury risk-free. The trunk may be extended between the bars of a protective barrier. Hands and arms extended through a barrier may be grasped or pressed against the barrier.

Elephants may be trained to target. The handler uses a long pole to touch a place where the elephant should go to or an area of the body that should be presented to the protective barrier for inspection or a procedure. Whistles or clickers may be used as a bridge for reinforcement.

### Handling in a Restraint Device

Handling an elephant in a restraint device prevents the elephant from moving away from the activity. There are many varieties of ERDs, from simple narrow aisles that limit side-to-side movement to elaborate units with movable walls and gates controlled manually, electrically, or hydraulically (Figs. 8.11, 8.12, 8.13, 8.14). The pipes and bars—in fact the entire construction—must be designed for the largest elephant that may be anticipated using the ERD. The American Aquarium and Zoo Association (AZA) encourages any zoo that maintains elephants to have an ERD. If mature bulls are to be exhibited, an ERD is a requirement. Currently there are over 50 North American zoos that have ERDs.[36,38,39,41]

Some ERDs are capable of restricting only lateral movement, and others are designed to tilt the elephant onto its side. It is important that the elephant is properly trained to enter the ERD and that the experience is positive or it may refuse to reenter the device. The concept of an ERD has been utilized in Asia for more than 1,000 years, and is still being used today. Instead of metal pipe, the Asian chute is constructed of upright poles buried in the ground like fence posts, heavy horizontal logs lashed together, and the whole structure anchored to a large tree (see figure 5.2).[14,45]

In spite of the many variations and designs of ERDs, all should have certain basic elements:

1. Must allow access to all four feet and legs, tusks, trunk, face, ears, both sides, hindquarters, and back.

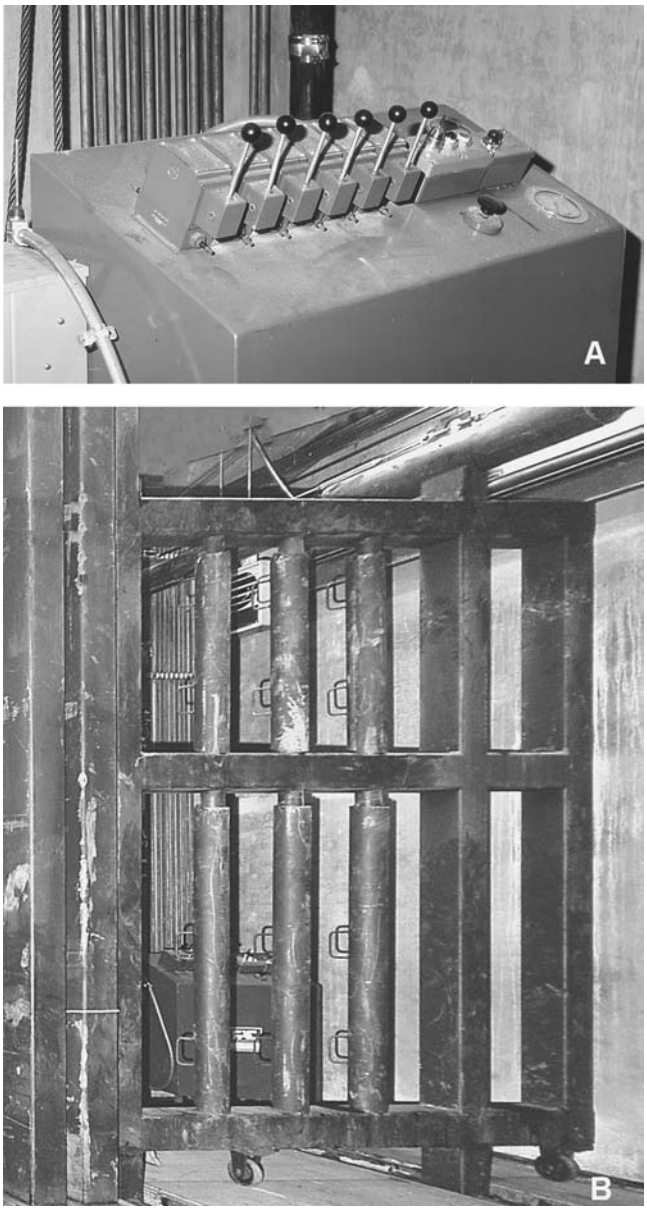


***Figure 8.11.*** *Movable wall on the first elephant restraint device built in a zoo in the United States. A) control panel, B) movable wall.*

2. Must be easily and quickly opened to free an elephant that has collapsed or has tried to lie down.
3. Must be able to contain an elephant comfortably for an extended time for prolonged medical or husbandry procedures.
4. Must be able to accommodate the largest and smallest elephants in the facility safely.

Additionally the ERD should be placed in an aisle so that all the elephants must pass through the ERD as a part of their daily routine. It is important that elephants are not confined in an ERD to undergo only unpleasant experiences. The ERD should be located in an area of the holding facility where it can be used any day and in any

***Figure 8.12.*** *An elephant restraint device (ERD) in a California zoo.*

***Figure 8.13.*** *End view of an ERD in a Florida zoo.*

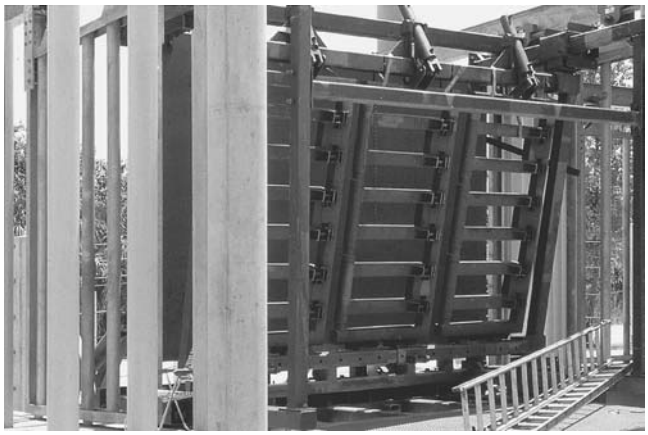

***Figure 8.14.*** *Side view of an ERD in a Florida zoo.*

kind of weather. While an elephant is confined to the ERD there should be a bypass to yards and enclosures for other elephants.

Accustoming elephants to accept an ERD requires considerable training. The trainer/handlers must know the individual elephant and the operation of the ERD. The ERD is a tool used to complement a sound elephant management program, and it should never be used as substitute for a poor management program or poorly trained staff.[25]

### No Contact

No contact is not an acceptable management strategy for elephants. Untrained elephants may resist any type of handling and must be sedated or anesthetized to conduct even the smallest nonpainful procedure. See Chapter 9.

## FOOT CARE

Proper foot care necessitates that an elephant be trained to lift a leg and hold the leg still for daily inspection of the sole, to place the foot on a tub or spindle, or to present a foot through a protective barrier.

Traveling elephants are of necessity handled directly. They are trained superbly and are generally easy to manage for veterinary procedures.[16] Traveling elephants may become closely attached to one handler/trainer, making them a problem when that person is unavailable to work with them.

Human injuries and deaths have been caused by traveling elephants that became aggressive for any number of reasons.[21,44] In some cases, owner/trainer/handlers did not always recognize musth and its ramifications, resulting in euthanasia.

## HANDLING THE RECUMBENT ELEPHANT

An elephant that is unable to arise from the recumbent position is difficult to deal with. The animal may refuse to try because of an injury or pain. One elephant known by the author was immobilized for a supposed dental problem but couldn't get up. Finally, she was diagnosed to have tetanus. She was lifted to her feet with a crane and treated with tetanus antitoxin, antibiotics, and tranquilizers, and she recovered.

An adult elephant may be turned from one side to the other using a *parbuckle* (see Chapter 9). An unconscious elephant should never be left lying in the sternal position because pressure on the abdomen prevents movement of the diaphragm and breathing.

### Slinging[9,26,31]

If it is necessary to place a sling on a recumbent elephant, a length of iron reinforcing rod used in construction may be pushed beneath the animal just behind the foreleg and just in front of the rear leg. Then a small rope is attached to the rod, pulled back in the opposite direction, attached to the appropriate sling straps (cargo bands), and pulled through. One strap behind the front limbs and another in front of the hindlimbs provides adequate lift. A safety strap should unite the front strap on one side around the chest to the other side (Fig. 8.15). Similarly, a butt strap should connect the rear straps. These may be anchored with ropes. They are used in case the elephant struggles or is tilted, creating a risk of the elephant slipping out of the sling. Both the front and rear sling straps should be attached to the crane hook, or an appropriate spacer may be used to keep the front and rear straps apart (Fig. 8.16). The crane operator should be provided with the weight of the elephant so the maximum angle can be calculated for the boom of the crane to lift the elephant safely. Sling straps are usually provided by the crane company.

### Commercial Sling[11]

An elephant weighing 2817 kg (6210 lb) required surgery on a foot, but she couldn't be laid down to administer an anesthetics agent or be chemically immobilized without causing potential damage to an arthritic elbow. A special sling and hoisting system was developed for this elephant so that she could be anesthetized while standing and then gently lowered to the floor. It was not possible to move a crane of sufficient capacity into the elephant house to support her. A suspension system and a hoist were designed and installed by local firms (suspension system: Columbia Wire and Iron Works, 555 N. Channel, Portland, Oregon 97217; hoist: Allied Power Products, Inc., Beaverton, Oregon, owner Robert Petterson). The suspension system consisted of two steel I beams braced on the top with right angle and diagonal steel strips bolted to the I beams, Fig. 8.17. The beams spanned a distance of 9.8 meters (32.2 feet) and were set on brackets anchored to solid concrete walls with multiple 19 mm by 150 mm (3/4 inch by 6 inch) epoxy anchor bolts.

The hoist system was powered by the hydraulic

***Figure 8.15.*** *Straps attached to a recumbent elephant.*

***Figure 8.16.*** *Lifting an elephant from the recumbent position with a crane.*

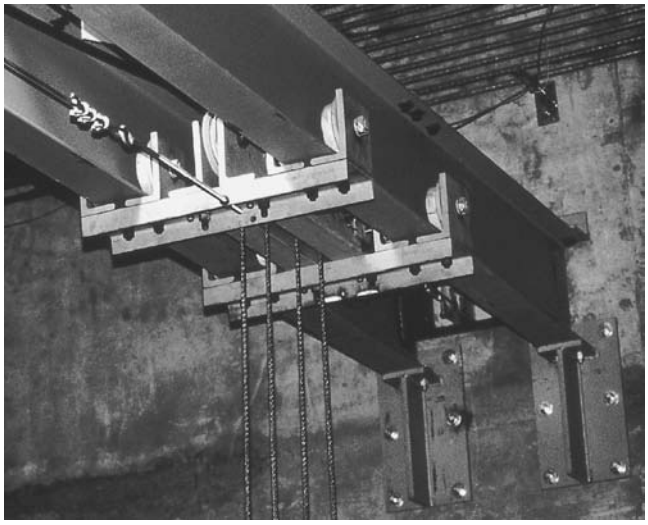

***Figure 8.17.*** *Hoist and trolley arrangement to lift and lower an anesthetized adult elephant.*

pump used to operate the door system in the elephant house. The hoist was designed to move horizontally on a "hammerhead" trolley, rolling on the suspension system's I beams. A continuous loop of wire cable extended from one wall to the other under the control of an electro/hydraulic diverter valve.

Vertical movement was achieved by a swivel hook/load block (with two sheaves), having a lifting capacity at the hook of 9072 kg (20,000 lbs). The vertical speed of the hoist, under load, was 0.3 meter (1 foot) per 3 seconds. Thus, it required approximately a minute to lower the elephant from the standing position to lateral recumbency.

Speed of descent and lifting was an important issue. After the elephant had collapsed into the sling, it was imperative that she be lowered into lateral recumbency as quickly as possible. An elephant has a unique thoracic anatomy. The pleural space is obliterated by fibrous tissue; thus, no negative pressure is possible. Inspiration is accomplished by flattening the dome of the diaphragm and by lateral movement of the rib cage. The lowest point on the ventral abdomen is caudal to the rib cage. When pressure is exerted on the abdomen, such as in a sling or sternal recumbency, the abdominal viscera are deflected cranially, putting pressure on the diaphragm. The sling also restricts lateral movement of the rib cage.

The sling design was adapted from a sling used to support horses with orthopedic challenges or rescuing horses by means of a helicopter. The sling bed (heavy nylon fabric) was supported by 12.2 inch (5.08 cm) nylon straps, each having a breaking strength of over

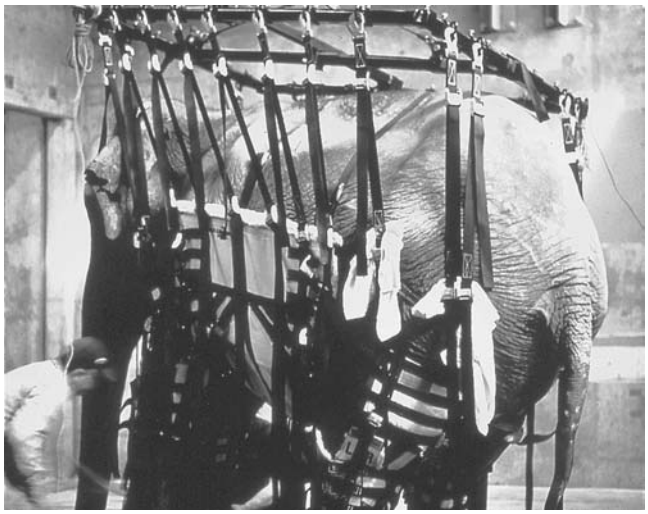

***Figure 8.18.*** *Sling and straps attached to an elephant for standing anesthesia and lowering to the recumbent position.*

***Figure 8.19.*** *Lifting an elephant off its feet following standing anesthesia.*

12,000 pounds (5443.2 kilograms) (Figs. 8.18, 8.19). The frame superstructure of the sling was constructed of square, metal-welded tubing with a wall thickness of 1/4 inch (0.64 cm) and outside dimension of 2 inches (5 cm).

The sling was designed for use with four hydraulic rams to adjust the fit of the sling to the animal. The frame was 44 inches (1.12 m) long and 29 inches (0.74 m) wide, with 22 eye rings to attach straps. Because of restricted lifting clearance in the elephant barn, it was not possible to use the hydraulic rams. Four choker cables were used to attach the metal frame to the hook on the hoist.

## TRANSPORTING ELEPHANTS

Transporting may be as simple as walking a trained elephant to a new enclosure, but if the elephant has been handled only from behind a barrier, the operation may be difficult. If two other trained elephants are on site, the untrained elephant may be tethered between them, or the elephant may be tethered between two heavy construction vehicles (Kollias 1993).[18] In one situation, a female Asian elephant was tethered between a large truck and a huge tractor and then walked to a railroad siding where she was loaded into a railroad boxcar that had been specially fitted to transport her from Denmark to Switzerland.

Circuses have been transporting trained elephants for decades. Special railroad cars or trucks are employed. Although ramps are usually used to load and off-load, adult elephants are capable of stepping down from a bed that is 0.8 m above the ground (Fig. 8.20). Transporting untrained zoo elephants may be an entirely different story; in fact, considerable effort must be made to acclimate the elephant to a crate or a simulated trailer such aa the trailer that would be used to transport the animal. In essence, the elephant must be trained to go into a crate and accept the confinement.

***Figure 8.20.*** *Elephant stepping down from a transport trailer.*

The author's experience may illustrate the challenge. An adult female Asian elephant was to be moved to another facility for breeding. She was trained to lead and

was a docile animal but would not voluntarily climb a ramp and enter the trailer. She was tethered on both front legs, the tethers were extended up through the trailer, and slack was taken up each time the elephant was coaxed to move ahead. Ultimately, she entered the trailer and was tethered in place.

She was to travel 724 km (450 miles), but approximately 16 km (10 miles) into the trip a keeper, following behind in another vehicle, noticed that her trunk was protruding through the floor of the trailer. Upon inspection, it was observed that a hole had been smashed through the floor (5 cm [2 in] oak planks) by the elephant repeatedly dropping to her knees. A metal sheet had to be welded to the under carriage of the trailer to prevent her from protruding a leg and injuring herself. She was returned to the zoo and off-loaded. Later she was successfully moved to another zoo after being trained to enter the trailer voluntarily.

Young elephants may be crated and moved via truck, train, airplane, or ship if the crates are adequately constructed. Adult elephants require special facilities (Fig. 8.21). Semitrailer or railroad cars must be reinforced inside with 6.4 mm (1/4 in) sheet iron to withstand the tusks and butting of elephants. Rings must be anchored in the floor to tether both front and hind legs.

Tethers must be kept short, and access must be provided for handlers to tether and untether the elephant. Normally, elephants are moved from trucks on short ramps, or at docks where the elephant can walk out at truckbed-level. They can climb or step down a few feet if trained to do so.

Transportation standards for elephants in the United States are included in the Animal Welfare Act, United States Code, Title 7, Chapter 54. Subpart F, paragraphs 3.136 through 3.142. Regulations for care in transit, frequency of feeding, watering, handling, and responsibilities of carriers and intermediate handlers are specified. Those involved in moving elephants in the United States should be familiar with these guidelines.

Transport by air must conform to the regulations of the International Air Transport Association Live Animal Regulations.[2]

## Pre-Trip Considerations

Moving an elephant may be a huge logistic operation. It is critical that adequate time be available to plan the trip and bring together equipment, staff, permits, and training to the point that the move may be made with the least amount of stress on the elephant and caregivers. A protocol for preshipment health assessment used by the Species Survival Plan (SSP) of the AZA is included in Appendix 14, "Recommended Elephant Preshipment Guidelines (AZA/SSP)."

A commercial animal mover had been contracted to move an elephant across the United States. He had an appropriate fifth wheel trailer to move the elephant, but he had to rent a tractor. The elephant was loaded and

***Figure 8.21.*** *A steel-reinforced crate used to transport an adult elephant.*

the journey began. Some 5 miles into the trip the truck was required to go through a truck inspection station. The inspector found that the license for the rented tractor was not current and the brakes on the trailer were defective.

The elephant couldn't be off-loaded, but fortunately a mechanic was able to service the brakes; a call to the rental company managed to get the tractor license brought up to date. A potential disaster was avoided.

## Crate Training

Not all of the following suggestions are necessary for every animal. The appropriate crate should be placed within the enclosure so the elephant is able to explore and smell it. Open both ends of the crate so an elephant can pass through the crate. Later close one end. If possible feed the elephant within the crate or at least provide treats such as fruit or vegetables. Close the gate after the elephant is comfortable, but quickly open it. Gradually increase the amount of time the elephant is kept in the crate.

## Transporting Circus Elephants

Circus elephants are moved by tractor/trailer vehicles or trains. One large circus in the United States has specially constructed railroad cars that are used to transport elephants and other hoofed stock. These cars are attended by experienced caregivers who are alert to challenges that may arise. The cars can be heated or cooled. Water is carried on board and food may be provided. Waste is removed en route as necessary.

Although the cars are circus property, they are pulled

by locomotives on the lines being used. Passenger and freight trains are given priority, so elephants must be adequately maintained for many hours in some instances.

An independent study conducted to determine stress in elephants transported by truck and train concluded that there was no evidence of hyper- or hypothermia in the elephants even during extreme weather conditions. Furthermore, levels of ammonia and carbon monoxide were below detectable levels at all times.[13,43]

## Transport in Range Countries

Elephants are moved within and between range countries to reduce a population that is overgrazing the forage in an area, reintroduce elephants to new areas, move elephants to public and private facilities, and occasionally for medical reasons.

Elephants may be transported directly from the capture site to another destination if the trip is less than 6 hours.[29] Generally this involves young crated elephants that must be tranquilized during transit. The use of long-acting tranquilizers has made such trips less traumatic to agitated elephants. These young animals have been immobilized to facilitate crating, and have been given an appropriate antidote after they are inside the crate. Renarcotization may occur, so the transportation team must be prepared to administer more antidote.

Establishing a correct dose for tranquilizing elephants during transport is an art as well as a science. Newcomers to elephant transport should contact someone with experience to avoid challenges such as an elephant becoming recumbent in a chute or crate, which may cause death.

The use of long-acting tranquilizers is relatively new (see Chapter 9). Haloperidol at 0.1 mg/kg administered intramuscularly has shown great promise. The effect of haloperidol may last for 20 hours. Other agents may last for weeks.

Vehicles used to transport young elephants should stop periodically, especially early in the journey to check for renarcotization (give more antidote), and later in case the elephant becomes more agitated, requiring more tranquilizer.

Newly captured adult elephants should be moved quickly to a *boma* (elephant enclosure) constructed close to the capture site. The immobilized elephant is carefully winched on rollers or poles onto a low flatbed trailer and transported while immobilized and in lateral recumbency. When the boma is reached, the elephant is recovered and confined to the boma for sufficient time to quiet down and become accustomed to human care and feeding. If the elephant is to be crated, the same technique as described for zoo elephants should be used.

Adult wild elephants usually require tranquilization when transported for long distances. Similar operations are carried out both in Asia and Africa. Guidelines for the in situ Translocation of the African Elephant for Conservation Purposes are published by the International Union for Conservation of Nature (IUCN) 2005.

## Elephant Transport Maxims

Remember the following:

1. Make certain that the vehicle (truck, trailer, train)
   a. Is licensed to travel through the appropriate states or countries
   b. Has functioning tires, brakes and lights
   c. Provides adequate ventilation
2. Adequate time must be devoted to training the elephant to move into the crate or vehicle. This may take 6 weeks.
3. The crate, trailer, or train must be constructed to accommodate and fully contain the elephant to be moved.
4. When lifting a crate with a crane, make certain the capacity of the crane is sufficient and it is capable of moving the crate without tilting.
5. Elephants are intelligent and inquisitive and will test all locking devices. Keep them out of reach of the trunk or design them to be elephant proof.
6. When transporting young elephants a considerable distance in a crate, make certain the animal has space to lie down on its side and get back up.
7. Adult elephants are capable of turning around in a crate even if provided only sufficient room to stand.[29] They may sit on their rumps or stand on their head and swivel.
8. If it will be necessary to tranquilize or sedate an elephant during transport, it would be wise to presedate to establish a proper dose.
9. Elephants produce copious amounts of urine. Use an absorbent bedding, such as sand or sawdust.
10. Crates must be anchored securely to the transport vehicle. This is particularly important when young elephants are moved by air or on a ship.
11. Untrained elephants should be tranquilized during transport, but not to the level of causing recumbency.
12. Select a route of travel that provides the shortest travel time consistent with passable roads. Avoid large cities and mountainous regions if possible.
13. Vehicle drivers should be appropriately licensed and experienced at moving animals. They should move out slowly and be gentle with stops. They should avoid swerving and go easy on curves.
14. Make certain that appropriate permits accompany the elephant. It is the responsibility of the shipper to determine what permits and licenses are required. Contact all appropriate governmental agencies.
15. Communicate with the destination facility personnel so that they are aware of the arrival time and are prepared to off-load the elephant.
16. Seek the cooperation of local law enforcement agencies to keep the elephants safe.

## Elephant Handlers

In Asian countries, elephant handlers (*mahouts, oozies*) have been a family tradition for thousands of years. Sons followed in the footsteps of a father who knew no other way of life than to live with and care for his elephant. Those traditions are being lost. There is a current challenge to attract young men to become elephant handlers who are willing to endure the long hours and learning processes needed to care for and train an elephant. Attempts are being made to help train handlers and even certify them as having completed training.

The loss of human life associated with working with elephants prior to 1940 would be unacceptable by Western standards today. Circuses wanted bulls to parade because they were bigger and had tusks.[40] Many of these bulls became aggressive during their musth period; trainers didn't appreciate the early stages of musth and were not equipped to isolate the bull until musth was over. In the timber industry in Asia, bulls were selected because they were bigger, stronger, and had tusks that made them more desirable. In one case an elephant killed 19 people (mahouts, villagers, family members).

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# 9 Chemical Restraint and General Anesthesia

## Section I: Chemical Restraint

Murray E. Fowler and Susan K. Mikota

### INTRODUCTION

Chemical immobilization in elephants is not a new phenomenon. For centuries, native Africans used poison-tipped arrows to immobilize and kill elephants for food (see Chapter 33). The birth of modern wildlife chemical restraint occurred when the first commercial dart was introduced in 1953 to administer curarelike drugs (gallamine) that had been used for ages by South American natives for hunting.[33]

Various nicotine formulations were used for domestic animals, but their use in elephants was precluded by the large volume required.[18,43] Another important event in modern chemical immobilization occurred when the Palmer Co. (Palmer Cap-Chur Inc., Douglasville, Georgia 30133) introduced the Cap-Chur projector and syringes for commercial use in the 1950s. In the late 1950s African workers expanded the use of succinylcholine chloride to immobilize various wild ungulate species including the elephant, but they found that elephants required higher doses, which were sometimes lethal. A number of morphine derivatives were tried, but it wasn't until 1963 when etorphine HCl, a powerful narcotic, was synthesized that a suitable drug for elephant immobilization finally materialized.[36] Since that time numerous narcotic and nonnarcotic agents have been used in elephant management and research, but etorphine is still considered the prime immobilizing agent for elephants.

The veterinarian in charge of an elephant immobilization is responsible for the safety of personnel, the elephant, and the facilities. Plan! Plan! Plan! Assume that anything may go wrong and plan for the worst possible occurrence. When the author hears a neophyte immobilizer say "I've never had any problem during chemical immobilization," I suspect the individual hasn't immobilized many animals.

The use of chemical restraint is both an art and a science. The drugs have known specific pharmacologic effects, but the physiologic and psychologic state of the elephant affects the outcome.[6,100] Experience with the agents, equipment, and elephants is vital. If in doubt, contact a colleague with elephant experience. If all safety conditions can't be met, delay the immobilization until conditions are corrected.

#### Terminology

Many different terms are used in the literature to describe chemical immobilization and anesthesia. Often the word *anesthesia* is used when it is sedation or immobilization that is described. The definition of some terms used in this chapter follows.

**Agonist.** A drug that produces its effect by interacting with a specific receptor site in the central nervous system (e.g., morphine interacts with opioid receptor sites).

**Analeptic.** A drug that acts as a restorative or stimulates cardiac or respiratory function (doxapram).

**Analgesic.** A drug that abolishes pain without producing unconsciousness or sleep (may be topical, local, systemic).

**Anesthesia.** A state without sensation or loss of sensation, with an accompanying reversible depression of nervous tissue, either locally or general.

**Anesthetic.** A drug or agent used to abolish the sense of pain and induce anesthesia.

**Antagonist.** An agent that counteracts or blocks the action of another agent, (in contrast with an antidote that neutralizes or counteracts the effects of a poison).

**Ataractic.** An agent capable of producing ataraxia (calmness); tranquilizers fit this classification.

**Ataraxia.** Impassiveness or calmness; perfect peace or calmness of mind; detached serenity without depression of mental faculties or clouding of consciousness.

**Catalepsy.** Prolonged maintenance of a fixed body position.

**Catatonia.** Muscular rigidity.

**Chemical immobilization.** To render an animal incapable of movement by the use of drugs.

**Dissociative anesthetic.** An agent that interrupts the association between the limbic and cortical systems producing analgesia and a catalepticlike state in which the eyes remain open and the swallowing reflex remains functional but there is dissociation from surroundings. Ketamine is an example.

**Narcosis.** A reversible state of central nervous system depression induced by a drug.

**Narcotic.** An agent that produces narcosis, but usually applied to agents having action similar to morphine (opiates).

**Neuroleptanesthesia.** A state of neuroleptoanalgesia and unconsciousness produced by the simultaneous administration of a narcotic analgesic and neuroleptic agent.

**Neuroleptic.** Any drug that favorably modifies psychotic clinical signs; the main categories of neuroleptics include the phenothiazines, butyrophenones, and thioxanthines.

**Neuroleptoanalgesia.** A state of quiescence, reduced awareness, and analgesia produced by the simultaneous administration of a neuroleptic agent and a narcotic analgesic.

**Reversal agent.** A drug that reverses the action of another drug.

**Sedation.** A mild degree of central nervous system depression, in which an animal is awake but calm, free of nervousness, and incapable of fully responding to external stimulation.

**Standing sedation.** A degree of central nervous system depression that enables the elephant to remain standing and calm.

**Tranquilization (ataraxia).** A state of calmness characterized by relaxation, reluctance to move, and potential indifference to minor pain.

**Tranquilizer.** A drug with a calming, soothing effect (neuroleptics, ataractics).

There is a continuum from analgesia (local or general) to tranquilization to sedation to immobilization to anesthesia (injectable or inhalant). It is not always clear when one action is supplanted by another, because the same agent may be used to produce varying effects depending on the dose and the physiologic and psychologic state of the elephant.

## DRUG DELIVERY[29]

### Handheld Syringe

Elephants may be trained to allow hand injections. It requires considerable force to thrust a needle through the thick skin of an elephant. Suitable sites for intramuscular injections are:

1. The triceps muscle just above the point of the elbow.
2. Thigh muscles at the back of the hind leg. The thinnest skin is on the medial aspect of the hind limbs, but access may be restricted.

With many wild animals the syringe is attached to the needle, and a quick thrust inserts the needle while at the same time the syringe plunger moves forward to expel the medication. This method is usually not practical in an elephant. It is preferable to inject elephants in two steps, first inserting the needle and then attaching the syringe. While holding the needle between the thumb and index finger, slap the injection site once or twice with the back of the hand and then quickly insert the needle. Attach the syringe and inject the drug.

It is important to use a needle of appropriate length when administering sedative or anesthetic agents. Most of the drugs used in elephants are intended to be delivered intramuscularly or intravenously. Inadvertent subcutaneous administration may alter the response. To be assured that the injection is given into muscle it may be preferable to use a 5 cm (2 in), or longer, spinal needle, especially on larger elephants.

### Stick Pole Syringe

Stick pole syringes (also called *pole syringes* or *jabstick*s) provide an extension of the arms, but the elephant may whirl, grasp the pole with the trunk, and destroy it. If the device is used in a protected contact situation, the elephant should be trained to accept the thrust. In one author's experience (SKM) a spring-loaded jabstick (Daninject, Børkop, Denmark) is effective. The speed of injection is rapid (<1 second); however, 10 ml is the maximum volume that may be administered at one time.

### Projected Darts

**Blow darts.** Blow darts (powered by a person blowing) are often used to immobilize or medicate captive wild animals, but this is not practical for use with elephants. However, compressed air may be used to project light plastic syringes (Telinject system), especially in young elephants.

**Powered weapons.** Palmer Cap-Chur projectors were the first commercially produced weapons for projecting darts. Now, numerous systems are available, utilizing carbon dioxide cartridges or percussion caps (powder charges) to supply the power to propel the dart. Many are available with telescopic sights. The reader is directed to other books for a review of details of types of equipment available.[29,62,67] See Appendix 3 for sources of equipment and supplies.

**Needles.** Needles for intramuscular injection in elephants should be stainless steel. For calves, 16 gauge (1.6 mm) outside diameter (OD), and 51 mm (2 in) long is recommended. For adults, using the handheld method, use 16 gauge (1.6 mm OD), 64 mm (2.5 in) long. For remote injection, 12 gauge (2.5 mm OD), 76–102 mm (3–4 in) long should be used.

Standard needle tips may cut a plug of skin, especially when projected by a powder charge.[82] Raath recommends that the tip of the needle be bent slightly toward the lumen of the needle to prevent this.[82] One of the authors (MEF) also suggests that holes be drilled in the shaft of the needle near the tip so that if the needle tip plugs, the contents may still be projected laterally.

An author's (MEF) preference is to have a welder experienced in welding stainless steel plug the tip. Then the tip should be filed to a point and side holes drilled for delivery of the drug. Needles for remote delivery should have a collar affixed to the shaft approximately midshaft. The larger needles must be specially ordered from veterinary equipment and supply sources. Cap-Chur needles for elephants are prefabricated with a closed tip and a side port.

### Sites for Intramuscular Administration of Agents

Although any muscle mass may be used, selection of an appropriate site for remote darting may depend on the position of the elephant in relation to the shooter and the thickness of the skin over a given area. From the rear, the muscles of the thigh (between the pelvic arch and the stifle) should be targeted. Be aware of the swinging of the tail that could deflect a dart. From the side, the lateral aspect of the thigh or the triceps muscle above the elbow of the front leg is recommended. An elephant may reach around and grasp the dart with the trunk. In order to make a perpendicular injection from a front angle, the cranial thigh region should be targeted. The gluteal muscles should be selected when darting from above (helicopter or high vantage point).

## STANDING SEDATION AND TRANQUILIZATION

Sedation may be used for procedures such as loading for transport, obtaining blood or other samples, specialized examination procedures such as ultrasound, artificial insemination, and others. The decision whether to use standing sedation (or full immobilization) depends on the procedure and potential discomfort, the temperament of the elephant, and considerations for staff safety.

In captive elephants, signs of impending sedation include slowing of ear and tail movement (as sedation deepens, movement ceases), sonorous breathing, protrusion of the penis from the prepuce, relaxation of the trunk, widening of the stance, and ataxia.

In free-ranging elephants, signs include slowing of the gait, lagging behind the herd, foot dragging, head weaving, stumbling, and lack of trunk coordination.

Even when a drug has been given for "standing" sedation, it is always possible that the elephant will become recumbent. Be prepared for that eventuality.

## PREPARATION FOR IMMOBILIZATION

Preplanning is the most important aspect of an immobilization procedure (planning, equipment, pads, hoist, safety issues, availability of emergency drugs for elephants and humans).

All contingencies should be well thought out. Nothing should be left to chance. It is wise to assume that untoward reactions may occur and to have drugs and syringes available to deal with emergencies.

Except for emergencies, captive elephants should fast for 24–48 hours prior to immobilization or anesthesia to minimize the amount of ingesta in the stomach and gas formation in the large intestine. Water should be withheld for 24 hours.

## INDUCTION OF IMMOBILIZATION

The safest method of immobilization is to administer the agent intravenously in a laterally recumbent elephant. However, not all agents are administered intravenously and some regimens call for preanesthetics. Immobilizing an adult elephant from a standing position is fraught with hazards. This is the only position available for free-ranging elephants, but in a zoo setting it is a real challenge to accomplish the task with complete safety.

As an example, consider the sequence following administration of etorphine intramuscularly. In 8–10 minutes the elephant stops conscious movement, the ears slow down and stop flapping. The trunk becomes relaxed and immobile. The elephant may become recumbent in several ways. It may simply collapse into sternal recumbency. If tethering allows it, the elephant may lose control of the hind legs and begin to slump back-

ward into a sitting posture before complete immobilization takes place. This is highly desirable and the least traumatic to the elephant.

A method of encouraging an elephant to sit down is to place a slip knot around the trunk near the mouth and, from a safe distance, have a couple of people pull on the rope. This procedure has the same effect as a snout rope on a pig. The elephant pulls against the rope and should sit when it loses control of the hind legs. The rope should never be anchored to a solid object as trauma to the trunk could result in nerve damage.

A more potentially traumatic way for an elephant to fall develops if an elephant starts to sway from side to side. It is now dangerous for any attendant to be near the elephant. If it sways too far it may step sideways to maintain balance. If it continues to sway it will overbalance and fall into lateral recumbency.

Unless veterinarians and attendants are able to attach ropes before the fall, as illustrated in Figure 9.1, selection of the up side may be a matter of chance. The time when ropes can be placed on a dangerous elephant is critical, and a knowledge of that time has to be gained by experience with the selected immobilizing agent. If in doubt, don't approach the elephant. Xylazine is notorious for producing what appears to be profound depression, but touch stimulation may cause an explosive arousal. If the elephant falls on the wrong side, it may be turned over using a parbuckle (Fig. 9.2).

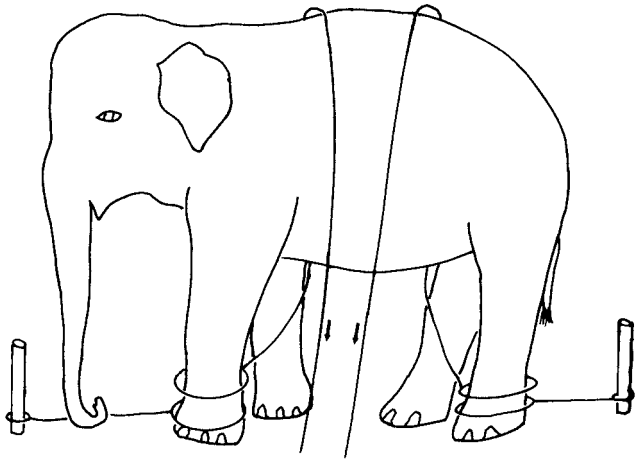

***Figure 9.1.*** *Diagram for placement of ropes to direct an elephant to lie down on the appropriate side during chemical immobilization.*

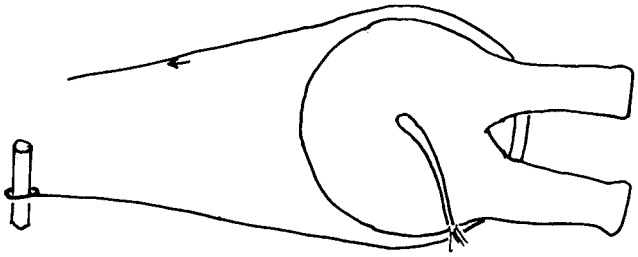

***Figure 9.2.*** *Diagram of the parbuckle being used to turn over an immobilized elephant.*

## COMPLICATIONS ASSOCIATED WITH CHEMICAL IMMOBILIZATION IN ELEPHANTS

### Overdose

An overdose may be caused by overestimating the weight of an elephant, miscalculation of the dose, or relying on memory for the dosage amount. It is not wise to rely on memory when dosing with these potent drugs. Rather, a small notebook listing drug dosages should be kept in the case containing the drugs and immobilizing equipment. Fortunately, most immobilizing drugs have reversal agents so if the overdose is recognized soon enough the overdose may be rectified.

An author (MEF) had not been involved in a clinical immobilization for a year when asked to sedate a giraffe with xylazine. The calculation was off by a decimal point and the giraffe began to collapse. Fortunately yohimbine was readily available and was administered intravenously in time to avert a disaster.

### Underdose

Underdosing may result from underestimating the weight of an elephant, failure to inject the full dose (by hand or remote dart), needle breakage without full injection, or depositing the drug in a poorly vascularized tissue (fascia, fat). Underdosing in a free-ranging situation is particularly hazardous. The elephant may fail to pass through an excitement phase quickly and continue to move away at a fast, incoordinated pace. Immobilization of free-ranging wild elephants usually requires a higher dose than for captive animals.

If a captive tame elephant is underdosed a supplemental dose may be administered, but this may be dangerous if the drug was deposited in a poorly vascularized tissue because the drug will continue to be absorbed, resulting in an eventual overdose. Also, if the elephant is truly underdosed and has become excited during the induction process, it may be hazardous for a person to approach the animal to administer a supplemental dose. A stick pole syringe or a spring-loaded jabstick may allow this to be accomplished. In either case, it may be better to stop the procedure, administer an antagonist, and try again another day.

### Encountering Hazards

In a zoo or private facility one must evaluate the site and minimize obstacles and hazards (moats, ponds, pools, falling close to a wall). Pools should be drained. Elephants should be tethered to control where they fall. An author (MEF) has sometimes unwisely accepted the boasts of keepers that they could control an elephant

without having to tether it. On one occasion, as the drug began to take effect, the keepers lost control and the elephant proceeded to walk into a deep pool and become recumbent. Two errors in judgment: 1) failing to tether the animal, and 2) not draining the pool beforehand. Concern that the elephant would drown when immobilization became more profound led to wading into the pool to administer the antagonist. Moats are another hazard in a zoo setting. Any tether used must be anchored to a substantial pillar or post.

When immobilizing free-ranging elephants one must be cognizant of the terrain to avoid recumbency in a narrow ravine or gorge. Immobilizations are best planned for cooler morning hours. Numerous factors, which are beyond the scope of this discussion, must be considered when immobilizing and translocating wild elephants. The reader is referred to the "IUCN/SSC AfESG Guidelines for the in situ Translocation of the African Elephant for Conservation Purposes."[24]

### Thermoregulatory Challenges

Hypothermia is a rare occurrence in an adult elephant; however, many of the immobilizing agents affect central thermoregulatory centers. Body temperature should be monitored during prolonged procedures.

Hyperthermia is more likely. Excessive muscular activity may cause an elevated body temperature. Elephants dissipate a portion of heat by flapping the ears, and this is one of the first reflexes lost when the elephant is immobilized. Although not described in elephants, some agents have produced a fatal malignant hyperthermia in other animals by disrupting the central control centers. If the body temperature begins to rise, cool water or ice packs placed on the ears and in the axillary or inguinal areas may help lower it. If it continues to rise (>38.9° C (102° F), the only alternative may be to reverse the narcosis and allow the elephant to begin cooling through its normal thermoregulatory processes.

### Hyper- and Hypotension

Narcotic agents produce hypertension, and alpha$_2$ adrenoceptor agents produce hypotension. Combinations of two of these agents smooth out blood pressure.

### Tachycardia

Heart rate should be monitored during any immobilization or anesthetic procedure. Tachycardia may be an indication that the depth of anesthesia is light.

### Respiratory Depression, Apnea

Respiratory depression will occur with an overdose of any of the immobilizing agents.

### Regurgitation

Prolonged immobilization with complete relaxation may allow stomach contents to enter the esophagus passively and be carried to the pharynx. Ideally, an elephant should be intubated to avoid inhalation of ingesta. With other animals positioning may be used to avoid inhalation, but adult elephants can not be positioned optimally.

### Falling Sternal

Recumbency in the sternal position will compromise respiration, and may result in death. Pull or push the elephant to lateral recumbency within no more than 15–20 minutes of recumbency.

### Falling with a Limb in a Malposition When Tethered

Depending on the tethering arrangement, the tether may cause the elephant to lie down with a leg under the body in an unnatural position.

### Fractured Tusks

Prominent tusks may be at risk of trauma during immobilization procedures. The least risk occurs if a tame zoo elephant can be directed to lateral recumbency for IV administration of the agent. The next most desirable preventative is to have padding ready to throw beneath the head (a sack of straw or foam pad), without placing a person in danger.

### Radial Paralysis from Prolonged Lateral Recumbency

Pulling the lower forelimb forward will minimize the risk. Padding, if possible, will help. A sign of radial paralysis is the elephant's inability to extend the affected limb forward in order to place weight on the limb. This may be evident when the animal first arises or occur a few minutes to an hour later when pressure from edema around the nerve disrupts impulse transmission.

### Miscellaneous Disorders Associated with Chemical Restraint

Contusions, exertional stress, exertional myopathy, cholinergic bradycardia, ventricular fibrillation, cardiac arrest, hemorrhage, acidosis, and hypoglycemia are not major problems in elephant immobilization, but veterinarians should be aware that any of these disorders may occur.[29] Fractures are rare, but the femur, pelvis, and metatarsal bones have been fractured during immobilization procedures.

Elephants have ruptured the stomach when falling during immobilization. Fasting for 24–48 hours will minimize the amount of ingesta in the gastrointestinal tract and decrease gas production, which could contribute to gastric rupture.

If an elephant is injected with an immobilizing agent and allowed to move without restraint, it may find a wall, fence, or other object to lean against. If the object is strong enough to support an elephant, sedation may deepen and the legs buckle to a folded position as the elephant slides down. Even though the elephant may

have been pulled into lateral recumbency, it may not be able to straighten the legs out in order to swing them to arise as it recovers.

A female elephant became recumbent with her back near a horizontal railroad rail fence. The rail was about 1.55 m (5 ft) above the ground. When she tried to arise, she couldn't get out from under the fence. Each time she rocked up she slipped further under the fence. She had to be physically pulled from under the fence.

The penis must be fully retracted into the prepuce before recovery is sufficient to release a wild elephant. In captivity the penis may remain protruded for an hour after the antagonist is administered, but if protrusion is prolonged the penis may become edematous and be subject to trauma. Spraying cold water on the extended penis may hasten retraction to just a few minutes.

### Human Injury

Personnel may be at risk during immobilization episodes. They may be accidentally exposed to the immobilizing agent or be traumatized in some way. Handlers should not be placed in dangerous positions. Always have antagonists for human use for the agent that is being used and have appropriately trained personnel for administering to a human.

### Estimation of Weight

Knowing the weight of an elephant is vital for medicating, establishing appropriate feed intake, or administration of chemical restraint agents. In captive zoo elephants, an actual weight should be established for each animal. Scales suitable for weighing elephants should be mandatory for institutions exhibiting elephants. Growing animals should be weighed at least every 6 months. Some institutions have installed platform scales in the elephant facility at a location that elephants routinely move through. In the United States, some zoos have requested that the state highway patrol bring in mobile truck scales, which allow weighing the forequarters and hindquarters separately. A formula is used to combine the two weights. The latter method requires a well-trained elephant that will stand on the scales on command in either free or protected contact.

For weight estimation in the field, see Chapter 7.[63]

## FAILURE OF SEDATION OR IMMOBILIZATION[29]

### Equipment Failure

High-impact darts may break at the needle hub, allowing the needle to embed deeply into the muscle mass. A syringe charge may fail to explode (faulty charge or insufficient force of impact to set off the charge). The propelling charge may be too low to carry the dart to the animal (an incorrect powder charge may have been selected, or a $CO_2$ cartridge might have been nearly empty).

Tail pieces should be symmetrical to prevent wobble. Air in the drug chamber may change the balance and hence affect the ballistics of the syringe. When loading syringes, excess space should be removed by adding an appropriate diluent to fill the chamber completely.

### Operator Fault

All equipment should be clean, lubricated, and in good repair. Needles should be inspected for plugs. The most common fault is for the operator to miss the target or to make an injection at an inappropriate site (fat, fascia). In captive elephants, avoid shooting at a moving target. Always try to have the syringe strike the target at a perpendicular angle to avoid ricochet and to produce a solid impact. When shooting from behind, tail switching must be considered. If the power charge for a Palmer syringe is inserted backward the charge will explode at the beginning of propulsion rather than at impact.

### Miscellaneous Conditions

Climate may have a bearing on successful immobilization. Wind may deflect a dart. Cold weather decreases the efficiency of $CO_2$ gas. A partially sedated elephant may stumble and fall into a precarious position or fracture a tusk or a bone. Access to ponds, pools, and moats should be prevented.

## LOCAL ANESTHESIA

In the adult elephant, infiltration of a surgical site with a local anesthetic agent is difficult because of the thickness of the skin. In young elephants or in areas of the body such as the trunk, prepuce, or vulva, local anesthesia may be administered.

Any of the standard local anesthetic agents are suitable (Table 9.1). Selection may depend on the rapidity of onset and duration of anesthesia desired or the experience of the operator. Any local anesthetic agent may be toxic if the administered dose is excessive. Some local anesthetic agents may be used topically on mucous membranes.

## ANALGESICS

Many immobilizing agents also have analgesic action. The primary use of strictly analgesic agents is not in the purview of chemical restraint and is discussed in Chapter 15.

## NARCOTIC IMMOBILIZING AGENTS

The immobilizing agents will be discussed individually. In most situations drug combinations are used to counteract undesirable pharmacologic action. The drugs and names used in range countries, the United States, and Europe are listed in Table 9.2.

**Table 9.1.** Local Anesthetic Agents

| Generic Name | Trade Name Source* | Duration of Anesthesia (Min) | Use | Signs of Toxicity |
|---|---|---|---|---|
| Procaine HCl | Novocaine[1]<br>1 or 2%<br>Generic[58] | 45–60 | Infiltration | Tremors, hyperexcitability, sweating |
| Chloroprocaine HCl | Nesacaine,<br>1 or 2%<br>Generic | 30–45 | Infiltration but not spinal | |
| Lidocaine HCl | Xylocaine,[46] 2%<br>Lignocaine<br>Generic | 60–120 | Infiltration, epidural, spinal, topical to mm | Death from ventricular fibrillation and cardiac arrest |
| Mepivacaine HCl | Carbocaine<br>Isocaine<br>Generic[1] | 90–180 | Infiltration, epidural | |
| Bupivacaine HCl | Marcaine[46]<br>Sensorcaine, 0.25% | 240–480 | Infiltration<br>Epidural | |
| Proparacaine HCl | Ophthaine,[69a] 0.5%<br>Alocaine[4a] | | Topical mm and ophthalmic | |
| Tetracaine HCl | Pontocaine,[63a] 0.5% | 60–80 | Topical mm and ophthalmic | Tearing, swelling, sensitivity to light, mydriasis |

mm = mucous membrane.
*Superscript number indicates a source listed in Appendix 3.

## Etorphine HCl (M99)

Etorphine used either singly or in combination with other agents is the drug of choice for immobilizing elephants.[2,7,20,21,23,25,26,27,29,31,39,47,34–38,44,51–60,62,,68,72,74,75,81,82,94,97–99,104]

**Pharmacology.** Etorphine is a highly potent narcotic analgesic, producing pharmacologic effects similar to those of morphine—namely, depression of the respiratory and cough centers, decreased gastrointestinal motility, elevated blood pressure,[39] tachycardia, and behavioral changes.[2,29]

Etorphine is a class II controlled drug in the United States. A current Drug Enforcement Administration (DEA) license is required for purchase and use. Additionally, the purchaser must be on an approved user list and must submit a completed DEA 222 form.[29]

**Indications for use.** Etorphine may be used in low doses to provide standing sedation or in higher doses for immobilization. When used as an anesthetic, it should be combined with other agents to counter its hypertensive effects. Etorphine and acepromazine (Immobilon) have been used to capture wild elephants[5] and for dealing with aggressive elephants.[11] Etorphine has been used extensively for minor and general surgery in captive elephants.

**Administration.** Etorphine may be administered IM or IV. Onset of immobilization occurs 5–15 minutes following an IM injection or 2–5 minutes if administered IV. If no antagonist is administered, recovery is slow, requiring up to 3 hours. When the antagonist is injected, the animal becomes ambulatory within 2–10 minutes. Etorphine should not be mixed with atropine because atropine reduces its solubility.

The speed of induction may be increased by adding hyaluronidase to the injected solution.[101] This is a special advantage when immobilizing free-ranging elephants.[45,69,70] The dosage of hyaluronidase ranges from 1500–4500 IU per dart.[59,60,73,75]

Etorphine has been used for prolonged anesthesia for up to 4 hours in captive elephants by intermittent administration of 1 mg etorphine IV every 15 minutes or by a continuous drip injection (4 mg etorphine in 250 ml of 0.9% sodium chloride solution) given at one drop per second.

**Side effects.** Respiratory depression results from inhibition of the respiratory centers in the brain and may influence acid base balance and the concentration of blood gasses.[44] Other effects include a decrease in gastrointestinal motility, which may result in intestinal tympany. Hypoxia, hypercapnea, and progressive metabolic acidosis may follow respiratory depression.[101] A portable ventilator may be readily constructed from two high-flow demand valves and the Y-piece of a large-animal anesthesia circuit to deliver intermittent positive pressure ventilation. The device has been shown to improve oxygenation dramatically and may be used in the field.[47] Oxygen saturation is easily monitored using pulse oximetry.[75] Monitoring is discussed further in "SECTION II: GENERAL ANESTHESIA."

Elephants, in contrast to other ungulates, do not experience an excitement phase following administration of etorphine.[101]

**Table 9.2.** Chemical Restraint Agents, Tranquilizers, and Neuroleptics Used in Elephant Management*

| | Trade (Commercial) Names | | | |
|---|---|---|---|---|
| Generic Name | North America | UK/Europe | Africa | India/SE Asia |
| **Sedatives and non-narcotic immobilizers** | | | | |
| Xylazine | Rompun[9]<br>Generic[72a] | Rompun[10] | Rompun,[11] xylazine powder | Rompun,[12]<br>Bomazine[64]<br>Xylazine |
| Detomidine | Dormosedan[53] | Domosedan[54] | Domosedan[16] | Dormosedan |
| Medetomidine HCl | Domitor[17,49a,76]<br>Meditomidine[53] (20 mg/ml[79]) | Domitor[49a] | Domitor[16]<br>Zylopine | |
| Ketamine HCl | Vetalar[22]<br>Ketaset[22]<br>Generic[72a]<br>Ketamine (200 mg/ml)[79] | Generic[10] | Anaket V[70]<br>Ketalar[52] | Generic |
| Tiletamine | Telazol[22] | Telazol[23] | Zoletil[50] | Zoletil |
| **Immobilizers, opiates** | | | | |
| Etorphine HCl | M99[76] Immobilon | M99[76] | M99[38] | M99, generic |
| Carfentanil Citrate | Wildnil[76]<br>Carfentanil (3mg/ml)[79] | Wildnil[67] | Wildnil[76] | Wildnil[76] |
| Sufentanil Citrate | Sufenta[33a] | N.A. | Sufental[35] | N.A |
| Meperidine HCl | Demerol generic,[1] | Generic | | |
| Butorphanol Tartrate | Torbugesic[22] | Torbugesic[23] | Torbugesic[7] | Torbugesic |
| **Benzodiazepines** | | | | |
| Diazepam | Valium[61a]<br>Generic[1] | Generic[2] | Valium[63] | Generic<br>Compose |
| Midazolam | Versed, generic[1,8a] | Generic | Dormicun[63] | N.A. |
| **Long-acting tranquilizers** | | | | |
| Haloperidol | Haldol[49b,62]<br>Haloperidol (20 mg/ml)[79] | | Serenace[67] | Serenace |
| Perphenazine enanthate | Trilafon [49a,65] | Trilifon[65] | Trilafon L.A.[65] | Trilafon[65] |
| Pipothiazine palmitate | Pipertil[44] | | Piportil Depot[44] | N.A. |
| Promethazine | | | Phenergan[44] | N.A. |
| Zuclophenthisol decanoate | N.A. | N.A. | Clopixol Depot[41] | N.A. |
| Zuclophenthisol acetate | N.A. | N.A. | Clopixol[41] acuphase[41] | N.A. |
| **Short-acting tranquilizers** | | | | |
| Acepromazine maleate | PromAce[22]<br>Generic[72a] | PromAce[23] | Combistress,[57] Neurotranq[38] | PromAce |
| Azaperone | Stresnil[43] | Stresnil[65] | Stresnil[30] | Stresnil |
| Chlorpromazine HCl | Thorazine[68] | Thorazine[68] | Largactil[44] | N.A. |
| Propionylpromazine | Combelen[9] | | Combelen[11] | |
| **Combination drugs** | | | | |
| M99/acepromazine | Immobilon | | Immobilon[32] | |
| Tiletamine/zolazepam | Telazol[22] | Telezol[23] | Zoletil[42] | Zoletil |
| Suxamethonium | N.A. | N.A. | Scoline[21] | N.A. |

N.A.= not available.
*There may be more than one source for some of these drugs. Numbers in superscript are sources, listed in Appendix 3.

Etorphine was administered to an African elephant at a German zoo. Instead of buckling on the hind legs, the front legs buckled and the elephant fell forward with the head between the front legs. His stiffened hind legs remained upright. The position obstructed air flow to the lungs and the elephant died before he could be pulled to lateral recumbency.[29]

Renarcotization (recycling) is seldom seen in elephants, but it may occur if multiple supplementary doses are given because it may be difficult to gauge the amount of an antagonist to use. Generally it is recommended to administer one-half the dose of the antagonist IV and the remainder IM.

Opioid narcotics elevate blood pressure and have been implicated in the etiology of a pink foam syndrome in wild African elephants. This emergency situation may be fatal. The syndrome manifests as pink froth from the trunk and is caused by pulmonary edema and capillary bleeding. Combining azaperone with opioid narcotics may counteract these hypertensive effects.[39,25,98]

Fatal hyperthermia may occur with etorphine administration. This is particularly a possibility when

small elephants have been chased with a helicopter or are highly excited at the time of injection. Body insulation and central thermoregulatory depression may inhibit heat dissipation. Always monitor core body temperature by inserting a clinical thermometer deep into the rectum with a gloved hand and arm. A string should be attached to the thermometer and clipped to the skin or some hair to avoid loss in the voluminous rectum and for periodic retrieval for reading.

Etorphine. Etorphine is extremely dangerous to humans. If injected accidentally, medical help should be sought immediately. The onset of sedation in humans is evident within 2 minutes following injection of a toxic dose and with the full effects within 5–10 minutes. The symptoms of a toxic injection in a person include loss of consciousness; slow, shallow respiration; cyanosis; miosis; weak pulse; fall in blood pressure; muscle twitching; and deep coma. Death is caused by respiratory failure.[101] Medical aid should be summoned immediately, but it may be crucial to administer the antagonist in the field. Naloxone (Narcan) is the drug of choice.[96] One ml of naloxone (0.4 mg/ml) should be administered IV, immediately followed by additional 1 ml injections every 2–4 minutes until the victim is under hospital management. Diprenorphine (M5050) may be used if naloxone is not available (double the injected dose of etorphine). Initiation of cardiopulmonary resuscitation may be necessary.[29]

Etorphine is readily absorbed through mucous membranes and may be absorbed through the skin. It is important to avoid inhalation, ingestion, or contamination of the skin, particularly of the hands, which might touch the mouth. A cardinal rule for those working with chemical immobilizing agents is that nothing should be placed in the mouth or lips except food and drink. A pen or pencil, syringe case, needle cap, or syringe should never be held between the teeth and lips, which in turn should never be touched with a finger or hand.

A second rule is to use rubber gloves and protective goggles when loading dart syringes with narcotics.

Finally, immobilization of an elephant with any opiate should never be done by an individual working alone. Two persons, each capable of administering an antagonist IV to a human and with knowledge of cardiopulmonary resuscitation should be a minimum.

Individuals and institutions working with dangerous narcotics should develop a detailed emergency protocol appropriate to their situation.[78]

**Dosage.** See Table 9.3.

**Reversal agents.** Diprenorphine (M50-50) is a specific antagonist for etorphine. See Table 9.4. The standard dose is double the mg amount of etorphine administered. Other narcotic antagonists include naltrexone, naloxone,[96] and nalmefene.

## Carfentanil HCl (Wildnil)

**Pharmacology.** Carfentanil is the most potent opiate known, and it has similar pharmacologic actions to those of etorphine. Carfentanil is the narcotic most frequently used in place of etorphine. Carfentanil is a Class II drug in the United States with the same restrictions as for etorphine.

**Indications.** Carfentanil may be used in any application for which etorphine is applicable.[49]

**Administration.** Carfentanil may be administered IV or IM. It is usually combined with xylazine or other alpha$_2$ adrenergic agonists or tranquilizers to smooth out induction and recovery.[29] The mean time to recumbency is 10.1 minutes with a mean duration of 73 minutes (R = 4–187 minutes). Supplemental doses are required after 33 minutes.

**Side effects.** Carfentanil has a wide margin of safety, with dosages ranging from 0.0006–0.00286 mg/kg. Side effects are similar to those of etorphine, but renarcotization is more common with carfentanil unless naltrexone is used as the antagonist. Two of seventeen elephants immobilized with carfentanil showed mild renarcotization 2–3 hours following reversal.[49] Signs included head pressing and continual lying down and getting up. Respiratory depression is rare.

**Reversal agents.** Naltrexone is the antagonist of choice, but diprenorphine (M50-50), naloxone, and nalmefene may also be used.

## Butorphanol Tartrate (Torbugesic)

**Pharmacology.** Butorphanol is a centrally acting, narcotic analgesic, with both agonist and antagonist actions.

**Indications.** Butorphanol has been used extensively in elephant clinical practice, either alone or in combination with other sedatives, to calm an excitable animal for examination or minor surgical procedures. In horses, it is used to alleviate abdominal pain caused by obstruction of the intestine, spasmodic colic, tympanitic colic, and postpartum pain.

**Administration.** Butorphanol may be administered IV, IM, or SC in a dosage range from 0.01–0.03 mg/kg. The drug is supplied in vials containing 10 mg/ml.

**Side effects.** Slight ataxia initially following IV administration may be seen.

**Reversal agents.** Butorphanol has narcotic antagonistic action approximately equivalent to that of nalorphine. A marked overdose of butorphanol may be reversed by a larger dose of nalorphine, because nalorphine has no agonistic effect.

**Table 9.3.** Dosages for Selected Chemical Restraint Drugs for Elephants*

| Agent Generic Name and Species Reported | Dosage (mg/kg) | Route | Comments | Ref |
|---|---|---|---|---|
| **Sedatives and non-narcotic immobilizers** | | | | |
| Ketamine | NA | NA | not used alone | |
| Detomidine (Asian) | 0.0055 | IM | | a |
| Medetomidine (Asian) | 0.003-0.005 | IM | | 27 |
| Xylazine | | | may cause bradycardia or 1st degree heart block | 86 |
| Xylazine (Asian; adult, sedation) | 0.04-0.08 | IM or slow IV | doses > 400 mg may cause recumbency | 29,84 |
| Xylazine (Asian; adult, immobilization) | 0.10-0.18 | IM or slow IV | | 29 |
| Xylazine (African; adult, sedation) | 0.08-0.10 | IM | best in combination w other drugs for juveniles | 29,46,60 |
| Xylazine (African; adult, immobilization) | 0.15-0.20 | IM | best in combination w other drugs for juveniles | 29 |
| **Immobilizers, opiates **** | | | | |
| Butorphanol (African) | 0.01-0.03 | IM | | 29,83 |
| Carfentanil (Asian; adult) | 0.002-0.004 | IM | Total dose 5-12 mg | 29,46,60 |
| Carfentanil (African; adult) | 0.0013-0.0024 | IM | Total dose 3-12 mg | 29,52,53,60 |
| Etorphine (Asian; adult) | 0.002-0.004 | IM | Total dose 5-20 | 11,31,68 |
| Etorphine (African; adult) | 0.0015-0.003 | IM | Total dose 6-20 | 29,52,60,62,74 |
| **Benzodiazepines** | | | | |
| Diazepam (Asian) | 400-800 mg total dose | IM | to control seizures | b |
| **Short-actng tranquilizers** | | | | |
| Acepromazine (Asian and African) | 0.004-0.06 | IM, IV | may cause photosensitization | 29 |
| Azaperone (Asian) | 0.024-0.038 | IM | | 93 |
| Azaperone (African) | 0.056-0.107 | IM, IV | | 81,83 |
| **Long-acting tranquilizers** | | | | |
| Haloperidol lactate (Asian) | 40-100 mg total dose | IM<br>PO (BID) | | 14 |
| Haloperidol lactate (African) | 40-120 mg total dose based on height | IM | do not give to juveniles < 1.6m; do not use decanoate formulation | 25 |
| Perphenazine enanthate (African) | 100-300 mg total dose | IM | | 25,81 |
| Perphenazine enanthate (Asian) | 200-250 mg total dose | IM | n=4 adult Asian elephants 1800-3800 kg | c |
| Zuclophenthixol acetate (Asian; adult) | 480 mg followed by 500 mg at 5 h | PO | single report | 92 |
| **Combination drugs** | | | | |
| Azaperone and Butorphanol (African) | Azap:0.12 mg/kg<br>But: 10 mg total dose | IM | single report | 83 |
| Telazol (Tiletamine and Zolazepam) (African) | 3.0 | IM | single report; recumbent in 2 min; recovered in 6 h | 62 |
| Xylazine and Acepromazine (Asian; immobilization) | Xyl: 0.12<br>Ace: 0.05 | IM | | 72 |
| Xylazine and Butorphanol (African; sedation) | Xyl:0.035-0.16<br>But:0.005-0.036 | IM or IV | Give Xyl IM and then But IV 20-40 minutes later or give together IV (at lower dose) | 44,83 |
| Xylazine and Ketamine (Asian adult; sedation) | Xyl:0.1<br>Ket:0.3-0.7 | IM | | 49 |
| Xylazine and Ketamine (Asian adult; immobilization) | Xyl:0.12<br>Ket:0.12 | IM | | 72 |
| Xylazine and Ketamine (Asian juvenile; immobilization) | Xyl:0.12<br>Ket:0.33 | IM | | 29 |
| Xylazine and Ketamine (African; sedation) | Xyl:0.1 ± 0.04<br>Ket:0.6 ± 0.13 | IM | | 44 |
| Xylazine and Ketamine (African adult; immobilization) | Xyl:0.20<br>Ket:1.0-1.5 | IM | | 3 |
| Xylazine and Ketamine (African juvenile; immobilization) | Xyl:0.14<br>Ket:1.14 | IM | | 29 |

a) deSilva,D.D.N, and Kuruwita,V.Y. 1994. Sedation of wild elephants using detomidine HCl in Sri Lanka, 5th Intl Congress of Veterinary Anesthesia p.6. b) personal communication, Dr. J Cheeran, India, 2001 c) Mikota, unpublished

* Dosages are presented as a guideline only. There are few controlled studies and sample sizes are limited. Doasges reported for one elephant species may or may not be applicable to the other species. Drug effects may differ bewteen adults and juveniles and between healthy and ill elephants, Free-ranging or excited elephants may require higher doses. Variation may occur between individuals. Consultation with an experienced elephant veterinarian for current recommendations is advisable. The attending veterinarian assumes responsibility for the use of this information. See the text and also The Elephant Formulary (www.elephantcare.org) for additional details.

** Opioid narcotics elevate blood pressure and may cause a fatal pink foam syndrome in wild African elephants. Combining with azaperone may counteract. See discussion in text.

**Table 9.4.** Antagonists used in elephant immobilization and anesthesia

| | Trade (Commercial) Names[a] | | | | |
|---|---|---|---|---|---|
| Generic Name | North America | UK/Europe | Africa | India/SE Asia | Dosage mg/kg or Total Dose (mg)[a] |
| **Narcotic antagonists** [b] | | | | | |
| Diprenorphine HCl | M50-50[76]<br>2 mg/ml Revivon | M5050[76] | M50-50[38] | M50-50 | 0.0083 ± 0.001 mg/kg [94] |
| Naloxone HCl | Narcan [18a,76]<br>0.4 mg/ml | Narcan[18a] | Narcan[76] | | 0.004 mg/kg [83]<br>10-50 mg total dose [15,91,96] |
| Nalorphine HBr | N.A. | Nalline[48] | Nalor-phine injection | | 0.3-0.9 mg/kg |
| Naltrexone HCl | Trexan[76]<br>Naltrexone 50 mg/ml[79] | Trexonil[76] | Trexan[76] | | 50-100 mg naltrexone:1 mg narcotic [47,57,60] |
| Nalmefene HCl | Nalmafene[37]<br>50 mg/ml | | | | 0.116-0.332 mg/kg [52,94] |
| **Alpha$_2$ adrenoceptor antagonists** | | | | | |
| Atipamezole HCl | Antisedan[49a,53]<br>5 mg/ml | Antisedan[49a] | Antisedan[16] | Antisedan | 1 mg Atip: 10 mg Xyl IM or slow IV [17,84] |
| Tolazoline HCl | Priscoline<br>Tolazine<br>200 mg/ml [40,79] | Tolazoline[40] | Priscoline[15] | | 0.5 mg/kg IV [3,81]<br>1 mg Tol: 1 mg Xyl IV (partial reversal) or 2mg Tol: 1 mg Xyl IV (full reversal)[c] |
| Yohimbine HCl | Antagonil[76]<br>Yobine[40]<br>Yohimbine 10 mg/ml[79] | | Antagonil[76] | Antagonil | 0.05-0.13 mg/kg IV [17,50,86] |

[a] The superscript numbers after the drug names refer to manufacturers/distributors listed in the appendix; the superscripts after the dosages refer to references.
[b] Narcotic antagonists may be given IV or the dose may be divided and administered partially IV and partially IM.
[c] personal communication, Dr. Dennis Schmitt, Springfield, MO, 2001

## NONNARCOTIC IMMOBILIZING AGENTS

### Xylazine HCL (Rompun)

**Pharmacology.** Xylazine is a nonnarcotic sedative, analgesic, and muscle relaxant, producing its effect by stimulating both central and peripheral presynaptic $\alpha_2$ adrenoceptors. Under the influence of xylazine, animals appear to be sleeping. Other actions include depressed thermoregulation (hypothermia); hyperglycemia; decreased heart rate, cardiac output, and aortic flow; temporary increase in blood pressure followed by hypotension; and respiratory depression.

**Indications for use.** Xylazine is used for sedation, muscle relaxation, immobilization, and analgesia.[14,17,29,50,53,66,77,84,86,88–92,101,103] Slightly painful or distressing procedures may be carried out, especially in untrustworthy elephants[48,88]; however, sudden arousal is possible with loud noises or other stimulation. Xylazine has been used to calm elephants that are afraid of crowds (ochlophobia) in India.[14] Bosi reports on the use of xylazine for the capture of wild elephants.[12] Xylazine potentiates the actions of tranquilizers, other nonnarcotic sedatives and opiates. It is frequently combined with other agents to provide a safer and more effective immobilization cocktail, and it allows a reduction in the indicated dosage of the other drugs in the cocktail.[101]

Xylazine has been recommended for tranquilization of elephants moved by air or rail,[10,13,14,19,65] but this may be dangerous because elephants tend to want to lie down with deep sedation. Other drugs such as azaperone may be more suitable. Aik recommends xylazine for training captured elephants.[1] Intravenous xylazine was administered to an Asian bull elephant in musth for treatment of a foot abscess. The dose was titrated from 0.033 to 0.072 mg/kg to provide standing sedation and yet retain response to vocal commands.[46] Partial reversal with atipamezole made the animal more responsive.

**Administration.** Xylazine is supplied in 20 mg/ml and 100 mg/ml solutions and may be given IV or IM at doses ranging from 0.08–0.1 mg/kg for standing sedation in captive elephants and 0.15–0.2 mg/kg for immobilization. For dosage, see Table 9.3. Ideally, the elephant should not be excited or apprehensive prior to administration of xylazine, and the elephant should be left undisturbed for 20 minutes following injection. If an elephant is highly excited or agitated it may require a 25–33% increase of the dose for a normal elephant.[88,89]

Elephants that are in poor condition, weak, sick, in advanced pregnancy, or exhausted from a long truck journey should receive a lower dose.[89]

Immobilization occurs within 3–5 minutes following IV injection or 10–15 minutes after IM injection. Analgesia lasts from 15–30 minutes, but the sleeplike state is maintained for 1–2 hours. Painful procedures should not be performed after 30 minutes.

Deep sedation is characterized by deep snoring respiration, salivation, flaccid trunk when standing, coiled

trunk position when lying down, protrusion of the penis, dilated pupils, and dribbling of urine. Profuse salivation may occur.

Two experiences may illustrate some of the challenges of xylazine sedation. An elephant was slated for radiographs of the tusk roots, but would not tolerate the placement of a cassette. A light dose of xylazine was administered IV. As is typical, when starting to lose muscle control, the elephant walked to the corner of a railroad rail fence. She put her mouth over the top rail and refused to move out of that position. It was necessary to reverse the sedation and repeat later with restraint that prevented her from wandering in order to gain access to the site to be radiographed.[29]

In another elephant, the standing sedation was satisfactory to accomplish the task. Reversal agents were not available at that time so the standing elephant was left under the observation of keepers. The author left the zoo but received a radio call to return to the zoo quickly because the elephant had fallen down and seemed oblivious to any stimulus to get up. When she was examined, she had normal parameters (heart rate, respiration, body temperature), but she was snoring soundly. She remained in lateral recumbency for 6 hours.

**Side effects.** Stimulation during the induction stage may prevent optimum sedation. Seemingly sedated animals have roused explosively, negating the sedation. If stimulation is discontinued and the elephant left undisturbed it may drift back into sedation. Occasionally, muscle tremors, bradycardia, and partial atrioventricular block occur with standard doses. Atropine (0.04 mg/kg) may be given to counter cardiac effects and diminish salivation. Intracarotid administration produces transient seizures and collapse.

Rarely, the opposite of the sedative effect will occur. A male elephant had been injected with a therapeutic dose of xylazine; 2.5 minutes later he tilted his head upward and stepped backward. In 4–5 minutes he trumpeted loudly and started to shake his head, which was followed by complete delirium. After 10 minutes of violent excitement, he started to calm down.[85] This appears to be like the effect of an intraarterial injection of xylazine that an author (MEF) has seen in llamas *Lama glama.*

One author (SKM) had two cases in Asian elephants where xylazine caused sudden recumbency when administered at a low dose typically used for standing sedation. Both elephants were lactating and this may have had some relationship.

Photosensitization has been noted when xylazine was administered in combination with ketamine.[13]

**Reversal agents.** Yohimbine HCl (0.125–0.25 mg/kg) reverses the effects of sedation in elephants, presumably by blocking alpha$_2$ adrenoceptors, as it does in experimental animals. See Table 9.4 for names and dosage.

Tolazoline may be administered IV at twice the dose of the xylazine administered, or 0.05 mg/kg IV.[3,60,81] Atipamezole is also effective when administered IM or slowly IV at 5–10 mg per 100 mg of xylazine. [13,46,64,84]

## Detomidine HCl (Dormosedan)

**Pharmacology.** Detomidine is a xylazine analog that is longer acting and more potent than xylazine. It is a nonnarcotic sedative and analgesic. Bradycardia, diuresis, and hypersalivation have been noted. High doses of detomidine produce deep sedation leading to a loss of consciousness and a light plane of anesthesia.[101] The duration of effect may last for over 6 hours unless reversed.

**Indications.** Detomidine may be used in all situations where xylazine is appropriate.

**Administration.** Supplied in vials of 0.1 mg/ml, detomidine may be administered intravenously or intramuscularly.[101] The highest dose is necessary for analgesia. When administered IV, the onset is 2–4 minutes; when given IM, the onset is 3–5 minutes.

**Side effects.** Detomidine has the same side effects as xylazine (hypertension, bradycardia, sweating, and, rarely, excitement rather than sedation).

**Reversal agents.** The most satisfactory antagonist is atipamezole (see Table 9.4). It is available in Europe, Africa, Australia, Canada, Scandinavia, and the United States. Yohimbine and tolazoline may also be used.

## Medetomidine HCl (Dormitor)

**Pharmacology.** Medetomidine is the newest and most potent in the line of $\alpha_2$ adrenoreceptor agonists, with action more specifically on receptors associated with sedation and analgesia. Other pharmacologic effects include relief of anxiety, bradycardia, hypotension, and hypothermia.[29]

**Indications.** It is primarily used as an immobilizing agent in combination with other drugs.[87]

**Administration.** The drug is rarely used alone. Medetomidine may be administered IV, SQ, or IM at dosages of 0.04–0.08 mg/kg for standing sedation. Induction is 2–5 minutes. The duration of effect is 66–134 min. Snoring is a consistent sign of sedation.

**Side effects.** Medetomidine is one of the safest immobilizing agents available. Bradycardia is an inherent action of $\alpha_2$ agonists.

**Reversal agents.** Atipamezole is the specific antagonist.

### Ketamine HCl (Ketolar, Vetalar, Ketaject, Ketanest)

**Pharmacology.** Ketamine (a cyclohexamine compound) is a nonbarbiturate dissociative anesthetic agent. The animal usually retains normal pharyngeal-laryngeal reflexes. This desirable effect minimizes accidental aspiration of food or ingesta. However, endotracheal intubation is difficult when ketamine is the only agent used. Nystagmus may be noted during induction.

Ketamine crosses the placenta in all species so the fetus is sedated. Ketamine is not known to produce abortion.

Ketamine produces an increased respiratory rate with a decrease in the tidal volume. If ketamine is given intravenously at a too-rapid rate, apnea may be produced.

Ketamine does not produce skeletal muscle relaxation; rather, it produces catatonia. There is profound analgesia at medium to high dosages, although analgesia of the visceral peritoneum may be less than optimal. Excessive salivation may be alleviated with atropine.

Ketamine produces a fixed expression in the eyes. The eyelids stay open, and yet the cornea usually remains moist. It should be routine practice to instill an ophthalmic ointment into the conjunctival sac. The eyes should be protected from direct sunlight. Occasionally, corneal ulceration has resulted from prolonged exposure. Palpebral reflexes persist. Ketamine is detoxified in the liver, and metabolites are excreted via the urine.

**Indications.** Ketamine should not be used as a sole immobilizing or anesthetic agent in elephants. It should be combined with a tranquilizer or one of the alpha$_2$ agonists.[4]

**Administration.** Ketamine is supplied as a solution in 100 mg/ml and 200 mg/ml concentrations. It may be administered IM or IV. In a captive situation, when combinations are used, the tranquilizer or alpha$_2$ agonist should be administered 15–20 minutes before the ketamine because ketamine action is much more rapid (3–5 minutes) than that of other agents. Giving the drug with the longer induction time first helps to prevent the catatonia typical of ketamine.

**Side effects.** Tonic-clonic convulsions are produced in some species but have not been observed in elephants. Should convulsions occur, diazepam or midazolam should be administered and not other tranquilizers. Ketamine should never be used as the sole immobilizing or anesthetic agent in elephants. Side effects are generally ameliorated when the combined drug is administered.

Many animals experience transitory pain when ketamine is injected IM. Supplemental ketamine should be administered IV because the IM route fosters absorption into fat resulting in a prolonged recovery.

**Reversal agents.** There is no known clinical antagonist for ketamine; however, yohimbine and tolazoline have been administered.[101]

### Tiletamine HCl and Zolazepam HCl (Telazol—Fort Dodge; Zoletil—Palmvet, South Africa and Europe)

**Pharmacology.** Telazol is a combination of equal parts by weight of tiletamine base (an analog of ketamine) and zolazepam, a diazepinone tranquilizer related to diazepam. The combination produces the rapid dissociative anesthesia typical of ketamine, but with the relaxing action of zolazepam.

**Indications.** Telazol may be indicated for use in captive elephant calves, but the volume of solution required for an adult would be prohibitive. A combination of medetomidine and ketamine would be a better choice for adults. In practice, Telazol functions similarly to a combination of xylazine and ketamine.

**Administration.** The 5 ml vial contains 50 mg/ml of tiletamine base and 50 mg/ml of zolazepam. The administered dose is calculated from the combined (100 mg/ml) activity. Characteristically, the higher the dose, the longer the recovery period. There is only a single report of its use in elephants at a dose of 3 mg/kg. The elephant became recumbent in 2 minutes and recovered in 6 hours.[62]

**Side effects.** Side effects are similar to those seen with ketamine.

**Reversal agents.** No antagonist exists for tiletamine. Zolazepam effects may be reversed with flumazenil (Mazicon) (see Table 9.4).

## SHORT-ACTING TRANQUILIZERS[8,29,101]

### Acepromazine Maleate

**Pharmacology.** Acepromazine maleate is a potent tranquilizing agent that depresses the CNS. It produces muscular relaxation and reduces spontaneous activity. It is an antiemetic, and has hypotensive and hypothermic properties.

**Indications.** Acepromazine maleate is rarely used singly. When combined with opiates or cyclohexamines it has synergistic activity, lessening undesirable side effects, shortening induction time, and reducing the dose of the immobilizing agent needed.[101] Its muscle-relaxing characteristic is of particular value when combined with ketamine.

**Administration.** Acepromazine is supplied in a 10 mg/ml concentration that may be administered IV, IM, or SC. When given intravenously, effects are noted

within 1–3 minutes. Intramuscularly, 15–25 minutes are required for full effect.

**Side effects.** Acepromazine should be used cautiously in combination with other hypotensive agents. Occasionally, instead of producing CNS depression, it acts as a stimulant, and hyperexcitability ensues. Acepromazine is a phenothiazine derivative and may potentiate the toxicity of organophosphate parasiticides, so inquiry should be made about the prior use of these products before administration. Acepromazine is contraindicated for the control of convulsions in progress, because although the drug may prevent convulsions, it also reduces the threshold for convulsion stimuli already begun.

Acepromazine is a muscle relaxant, and one of the first signs of tranquilization is the protrusion of the penis from the prepuce. The elephant penis is a large organ and it is theoretically possible for an elephant to step on the penis.

**Reversal agents.** No antagonists are known.

### Chlorpromazine HCl (Thorazine, Largactil)

Chlorpromazine was one of the early phenothiazine derivative tranquilizers. It was used to control musth in bulls in Asia,[71] but is no longer used for elephant tranquilization because there are other agents that are safer and more effective.

**Side effects.** If chlorpromazine is accidentally administered intraarterially it causes an arteriolitis and potential necrosis of tissue supplied by the artery. It is not uncommon for an artery to be cannulated while attempting to administer medication in an ear vein.

An oral dose of 4.16 mg/kg caused severe depression and ultimate death of an elephant.

**Reversal agents.** No antagonists are known.

### Azaperone (Stresnil)

**Pharmacology.** Azaperone is a butyrophenone derivative producing sedation, decreased aggression, hypotension, decreased heart rate, and inhibition of ejaculation. It also has antiemetic action and may cause catalepsy at high doses.[101]

**Indications.** Azaperone is one of the newer short-acting tranquilizers. It has a wide margin of safety and has been used to tranquilize elephants as a single agent, but it is more commonly administered in combination with opiates or other anesthetic agents to smooth out immobilization and allow the use of lower doses of the primary agent.[83,95] It has been used for tusk trimming, minor surgery such as debridement of wounds, foot trimming, to decrease aggression with herdmates, and for control of musth.[93]

**Administration.** Azaperone may be administered IM or IV. The initial response may be observed in 15 minutes, with maximum effect in 15–25 minutes. The duration of effect is 2–3 hours. Signs of sedation include stupor, snoring, unwillingness to move, relaxed penis, and most importantly, no tendency or desire to lie down. See Table 9.3 for dosages.

**Side effects.** Hypotension is related to dosage, but it does not have an adverse effect on heart rate, cardiac output, or aortic blood flow.[101] Hallucinatory behavior has been noted when a sedated elephant was subjected to a mild stimulus.[93] Catalepsy may occur at high doses. Treatment of an overdose is symptomatic and supportive.

**Reversal agent.** No antagonists are known.

### Propionylpromazine HCl (Combelen)

**Pharmacology.** Propionylpromazine is a phenothiazine derivative with actions similar to those of acepromazine.[4]

**Indications.** Propionylpromazine is usually used in combination with opiates or cyclohexamines. It may also be used for tranquilization of elephants to be transported for short distances.

**Administration.** Propionylpromazine may be administered IM or IV. No doses are reported for elephants, but in other species the dose range is 0.03–0.2 mg/kg.

**Side effects.** Side effects include hypotension and excessive sedation if overdosed.

**Reversal agent.** No antagonists are known.

### Diazepam HCl (Valium, Tranimal, Tranimul)

**Pharmacology.** Diazepam acts on the thalamus and hypothalamus, inducing calm behavior. It has no peripheral autonomic blocking action, unlike some other tranquilizers. Transient ataxia may develop with higher doses as muscle relaxation progresses. Spinal reflexes are blocked. Diazepam is an effective anticonvulsant.

**Indications.** Diazepam prevents the convulsive effect of ketamine. If injected intravenously, it effectively controls convulsive seizures in progress. It can also be used as preanesthetic medication to calm an excited animal, and it may be used to control musth.[102]

**Administration.** Diazepam is supplied in solution in a concentration of 5 mg/ml and administered at a dose of 0.1–0.5 mg/kg. Onset is within 1–2 minutes when given intravenously. If given intramuscularly, it takes effect in 15–30 minutes, depending on the dose. Diazepam is metabolized slowly in the normal liver. Usually, clinical effects disappear within 60–90 minutes.

**Table 9.5.** Long-Acting Tranquilizers: Source, Onset, Duration, Dosage

| Agent | | Concentration of Solution | Onset of Action | Duration of Effect |
|---|---|---|---|---|
| Generic Name | Trade Name/Source | | | |
| Halperidol lactate | Haldol,[79] Serenace[67] | | 5–10 min | 8–18 hr |
| Perphenazine enanthate | Trilafon[65] | 100 mg/ml | 12–16 hr | 7–14 d |
| Pipothiazine palmitate | Piportil[44] | 50 mg/ml in sesame oil | 48–72 hr | 21–28 d |
| Zuclopenthixol acetate | Clopixol[41] | | 1 hr | 3–4 d |

**Side effects.** Diazepam may be chemically incompatible with other immobilizing agents and should not be mixed with them in the same syringe or in IV solutions. Some pain is associated with IM injection, and a transient inflammatory reaction may develop at the site.

**Reversal agents.** Flumazenil (Mazicon) is a specific antagonist for benzodiazepine tranquilizers. The dosage is 10 times the administered dose of diazepam or zolazepam.

## LONG-ACTING TRANQUILIZERS

See also Table 9.2.

### Haloperidol (Haldol, Serenace)

**Pharmacology.** Haloperidol is a butyrophenone derivative with actions similar to those of azaperone, but it is more potent and the effect lasts longer (8–18 hr).[101] It is available as a lactate, a decanoate (depot formulation), or as tablets. The decanoate form may cause severe anorexia and is not recommended (personal communication, Dr. H. Ebedes, South Africa, September 2001). Haloperidol lactate may be given to adult Asian elephants at a total dose of 40 mg IM or 40–100 mg PO BID (personal communication, Dr. Jacob Cheeran, Kerala, India, December 2000).

**Indications.** Haloperidol is used extensively as an antipsychotic in human medicine. It is indicated when transporting highly excitable or aggressive elephants on trips of short duration.[28]

**Administration.** Haloperidol may be administered IM or PO. For dosage and duration of action, see Table 9.5.

**Side effects.** In high doses, haloperidol may cause hypotension. It may also cause muscle tremors or rigidity, which may lead to hyperthermia. Diazepam will control tremors. Haloperidol decancate may decrease the appetite (personal communication, Dr. H. Ebedes, South Africa).

**Reversal agents.** No antagonists are known.

### Perphenazine Enanthate (Trilafon)

**Pharmacology.** Perphenazine is a phenothiazine derivative with a piperazine side chain. It has actions similar to other phenothiazine derivatives. Operant behavior is reduced; thus this agent would not be appropriate for training an elephant. Spontaneous motor activity is diminished and conditional avoidance behaviors are inhibited.

**Indications.** Perphenazine is used for sedation and calming of excitable or aggressive elephants, and for transporting them short distances.

**Administration.** Perphenazine should be injected only deep into a muscle mass because the product is in a sesame oil base. The drug may take 3 days to achieve an observable effect and may have a duration of 2-3 weeks. Haloperidol is sometimes used concurrently for transport situations. One of the authors (SKM) has administered perphenazine to four newly captured Asian elephants weighing 1800–3800 kg at total doses of 200–250 mg IM. All elephants exhibited a calming effect lasting about 2 weeks. Total doses of 100–300 mg have been used in wild African elephants.[25,27]

**Side effects.** Side effects are similar to those of acepromazine. Muscle tremors and convulsions may be seen at high doses.

**Reversal agent.** No antagonists are known.

### Pipothiazine Palmitate (Pipertil)

**Pharmacology.** Pipothiazine is a phenothiazine derivative with actions similar to those of other phenothiazine derivatives, but with prolonged action.[101]

**Indications.** Pipothiazine is used for prolonged tranquilization, for up to 28 days.

**Administration.** Pipothiazine should be administered only IM.

**Side effects.** Side effects are similar to perphenazine.

**Reversal agent.** No antagonists are known.

### Zuclophenthixol Decanoate (Clopixol Depot), Zuclophenthixol Acetate (Clopixol, Acuphase)

**Pharmacology.** Zuclophenthixol is a derivative of thioxanthene with actions similar to those of phenothiazine tranquilizers.[101]

**Indications.** Zuclophenthixol is used as an intermediate to long-acting tranquilizer. An Asian elephant (approximately 3750 kg) was given 480 mg of zuclopenthixol per os. An additional 400 mg was given 5 hours later and a vaginal vestibulotomy was performed under local anesthesia.[92]

**Reversal agent.** No antagonist is known.

### Promethazine HCl (Phenergan)

**Pharmacology.** Promethazine was developed as a long-acting antihistamine drug for humans, but it also has pronounced tranquilizing action. It has been used as a long-acting tranquilizer in elephants.

### Succinylcholine Chloride (Sucostrin, Anectine, Quelicin), Suxamethonium Chloride (Scoline)

**Pharmacology.** Succinylcholine chloride is a depolarizing muscle relaxant, with no analgesic or anesthetic properties.[29,40,41,42,61,79,80]

**Indications.** Succinylcholine was one of the first agents used for immobilizing elephants, but field workers noted the prolonged effects of the drug and concluded that it wasn't safe for use in elephants. It has been used to cull elephants.

**Administration.** Succinylcholine may be administered IM or IV.

**Side effects.** This drug paralyzes the muscles of respiration, and suffocation may ensue without assisted respiration.

**Reversal agents.** No antagonist is known. In most animals, the drug is metabolized rapidly, but this is not the case in elephants. Assisted respiration is the only way to maintain an animal, which is not a practical procedure in an elephant.

## IMMOBILIZATION SUPPORT DRUGS (Table 9.6)

### Atropine Sulfate

**Pharmacology.** Atropine is a parasympatholytic drug with action equivalent to blockage of the parasympathetic autonomic nervous system. It decreases salivation, sweating, gastrointestinal motility, bladder tone, and gastric and respiratory secretions. Vagal blockage produces tachycardia. Mydriasis occurs.

**Indications.** Atropine diminishes the excessive secretions induced by ketamine. It is also commonly used as a preanesthetic medication to prevent reflex vagal stimulation of the heart (cholinergic bradycardia) during induction.

**Administration.** Large-animal formulations are supplied as solutions at 15 mg/ml and small animal formulations at 0.5 mg/ml. Atropine may be given orally or

**Table 9.6.** Emergency and Support Drugs Used in Elephant Immobilization

| Drug Generic Name | Drug Trade Name Source* | Concentration | Indication | Dose (mg/kg) |
|---|---|---|---|---|
| Atropine sulfate | Atropine, generic[58] | 15 mg/ml | Bradycardia, antidote for organophosphate poisoning | 0.02–0.04 IV, IM, SQ |
| Epinephrine HCl | Adrenaline, epinephrine, generic[58] | 1:1000 (1 mg/ml) | Cardiac arrest, anaphylaxis** | Equine. 0.1, IV, IM |
| Doxapram HCl | Doxapram[22] | 20 mg/ml | Respiratory depression | Equine. 0.5–1.0 IV at 5 min intervals |
| Diazepam HCl | Valium[61a] | 5 mg/ml | Convulsions, catatonia | 0.1–0.2, IV |
| Lidocaine HCl | Xylocaine,[1] lidocaine generic[46] | 20 mg/ml | Ventricular arrhythmia | 0.25–0.5 IV, q 15 m. as needed |
| Sodium bicarbonate | Na bicarbonate, generic[58] | 1 mEq/ml | Metabolic acidosis | 0.5–1.0 mEq/kg, slowly, IV |
| Calcium gluconate or Ca borogluconate | Ca gluconate, generic[58] | 23%, 8 mEq/ml,Ca 10%, 0.43 mEq/ml | Hypocalcemia | 0.7 mEq/kg, slowly IV |
| Hyaluronidase | Wydase[78] | 150 NF units/ml | Enhance absorption of agents (Hoare 1999, Morton and Kock 1991) | |

*The superscript number refers to a drug manufacturer/distributor listed in Appendix 3.
**See discussion of anaphylaxis in Chapter 15.

parenterally at dosages of 0.04 mg/kg. Atropinization occurs within 1–15 minutes, depending on the route of administration.

**Side effects.** Dilated pupils should be protected from direct sunlight to prevent retinal damage. Atropine is contraindicated for patients with glaucoma. An Asian elephant became agitated following the IV administration of atropine (0.05 mg/kg) administered 90 minutes after azaperone was given.[32]

**Reversal agents.** Parasympathomimetic drugs may aid in counteracting the effects of atropine, but atropine is difficult to reverse.

## REVERSAL AGENTS

Antagonists are available for most chemical restraint agents (Table 9.4). Smooth recovery is the general rule with elephants, but the clinician must be aware of undesirable responses. One elephant failed to arise following administration of the antagonist. Subsequently it was determined that the elephant suffered from the early stages of tetanus and was unable to rise.

Renarcotization following the use of antagonists to reverse the effects of opiates has been reported in elephants. This was particularly true when multiple IM supplemental doses of the opiate were required to insure recumbency. The opiate agent may have a longer duration of action than the antagonist, or the agent may be injected into poorly vascularized tissue (fat, fascia), which may delay absorption beyond the duration of the antagonist's action.

## MAXIMS FOR CHEMICAL RESTRAINT OF ELEPHANTS

The following are maxims for elephant chemical restraint:

1. Time the immobilization to occur during appropriate ambient temperatures.
2. Avoid immobilizing in the afternoon so that prolonged recovery doesn't persist into the night.
3. It is inappropriate to leave a sedated elephant until it has recovered equilibrium and is able to use its trunk to eat and drink. This is particularly true if supplemental doses of an agent have been administered IM.
4. Absorption of drugs administered into fat, fascia or SC tissue is delayed. The initial response of the clinician may be to administer more, but this may be risky because the animal may become overdosed.
5. Avoid loud noises in the vicinity of an immobilization.
6. Fingers, syringes, or needle casings should never be placed in the mouth.
7. Eyes should be protected with glasses or goggles, and rubber gloves should be used when loading syringes.
8. First aid materials should be available at hand in case of injury or accidental human exposure to agents.
9. No one should work alone, especially when working with highly potent opiates. Written safety and emergency response protocols are advisable.

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# Section II: General Anesthesia

Eugene P. Steffey

## INTRODUCTION

Anesthetic management of an adult elephant for prolonged recumbency and major surgery is a formidable challenge. The massive size and destructive capabilities of the animal and mechanical considerations relative to recumbency and general anesthesia are intimidating and significant deterrents. Advances in technology, increased understanding of the elephant and its health care, and improved knowledge and skill in veterinary anesthesiology contribute to increasing incidence of successful anesthetic management under a wide range of circumstances and encourage our continued efforts.

The aim here is to provide a brief overview of the special and often unique characteristics of elephants, followed by a discussion of general principles and ultimately techniques of general anesthesia. Particular focus will be anesthetic management for procedures requiring prolonged recumbency (i.e., >1 hr) and surgical intervention—circumstances most closely aligned with the authors' clinical experience in various settings with captive animals.

## GENERAL CONSIDERATIONS

### Size

Elephants are the largest extant terrestrial animal. Animal size determines anesthetic equipment used (e.g., endotracheal tube and breathing circuit). Animal size also determines such considerations as animal positioning, unique anatomy and associated physiological adaptations, and often unknown or extreme pharmacological variants.

### Temperament

The behaviors of captive elephants encompass a spectrum of individuals that include the unapproachable, dangerous, and destructive (to people and facilities) types on one hand and the highly intelligent, affable, trained, and readily responsive (to handler commands) on the other. Generally as animal temperament in this spectrum worsens, circumstances facilitating ideal anesthetic conditions lessen, and the potential for important complications increases. In this regard a few examples include absolute or relative drug overdose, physical injury on induction and improper positioning during anesthesia, and recumbency with resultant likelihood for potentially life-threatening myopathy and/or neuropathy. On the other hand, working with a well-trained elephant and skilled staff in a species-friendly environment markedly improves the likelihood of successful health care delivery.

## PRINCIPLES OF ANESTHETIC MANAGEMENT OF ELEPHANTS

No one anesthetic technique is appropriate for all circumstances. It is best to determine specific needs and to apply an appropriately individualized plan. Considerations include characteristics of the specific patient, factors related to the procedure(s) necessitating anesthesia, the facilities available, and the personnel involved.

### Individual Characteristics

It is a distinct advantage if an elephant can be directed to lie down willingly at an appointed place. This individual likely permits appropriate positioning on a well-prepared body support surface and preanesthetic ve-

nous catheterization, which facilitates a smooth, atraumatic, injectable anesthetic induction. Contrast such an individual with the untrained captive, or most challenging, free-ranging individual.

## Procedural Factors

In comparison to traditional companion animal patients, the spectrum of procedures anticipated in an anesthetized elephant is quite limited. Procedures most commonly include management of dental disease, endoscopy, treatment of foot disease, ocular procedures, and reproductive considerations. Recumbency is limited to the lateral position.

## Facility Factors

Facilities related to the elephant's large size are often unique (e.g., specialized chutes or containments), limited to select locations, or must be adapted from existing conditions (e.g., use of a crane or hoisting device for movement and positioning of the recumbent patient) or constructed at the site (e.g., individualized sling and suspension system).[9] Specialized equipment must be available at the facility or obtained from vendors (e.g., a water bed)[9,10] most commonly supplying veterinarians engaged in equine or, more broadly, large-animal practice. Some existing large-animal equipment must be adapted further for use with elephants (e.g., rebreathing bag accommodations for the very large tidal volume of an adult African elephant or methods to facilitate mechanical ventilation).[18]

## Personnel Factors

Successfully delivered care to an ill or injured elephant requires the concerted efforts of a large number of individuals working directly or indirectly together as a team supervised by an overall health care leader. Anesthetic management is usually just one component of the overall program for care. The success of anesthetic management, though interdependent with other focused team efforts, depends on the competence of the anesthetist and support team; their collective knowledge and skills must be focused in both anesthesiology and the elephant.

## Pharmacology

**General principles.** The elephant's large size and behavior presents some unique pharmacological considerations. Usually exact weights to facilitate accurate drug dosing are not known. Except when administered by the best-trained individuals or when following chemical restraint, injectable drug administration is usually by intramuscular or subcutaneous routes, routes that are less accurate or predictable regarding time of drug onset, time to peak, and duration of effect. Little is known of the pharmacokinetics of selected drugs in elephants, so anesthetists must extrapolate knowledge about other species. Because in many initial circumstances, drugs are not administered intravenously, the drugs must be very potent (causing some potential for human harm via mistaken injection or absorption via mucous membranes) and/or very concentrated (beyond usual preparations distributed by pharmaceutical sources). These and other practical matters limit the number of drugs considered for sedation, chemical restraint, and general anesthesia.[7,8]

The most commonly used drugs for prolonged anesthetic management of recumbent elephants are classed as opioids and inhalation anesthetics.

**Opioids.** See "SECTION I: CHEMICAL RESTRAINT."

**Inhalation anesthetics.** Inhalation anesthetics have long been included in plans supporting required prolonged anesthetic management. Perhaps the earliest report was the use of trichlorethylene in a 25-year-old female Ceylon elephant to facilitate removal of a life-threatening pharyngeal foreign body (coconut).[24] Despite multiple examples of inhalation anesthesia since this early report, there are few reports of direct study of inhalation anesthetic action in elephants. Accordingly, most available information is compiled from case reports and anecdotal (often verbal) information or extrapolation from studies of other species.

Four volatile agents are currently available. Actions of three of these will be briefly highlighted: isoflurane, desflurane, and sevoflurane. Use of halothane for elephant anesthesia has been reported.[5,15,23,25,44] However, presently there is indication that its commercial availability worldwide will soon cease to exist. Accordingly, and because its clinical utility has been replaced by newer drugs with more desirable actions, halothane will not be discussed further. For information regarding halothane and more about the other contemporary anesthetics beyond that presented here readers are referred elsewhere.[38,39] In recent years, isoflurane has been most commonly noted in reports of elephant anesthesia,[4,5,9,10] and it is the agent with which the author has the most personal experience anesthetizing elephants. Background pharmacology of isoflurane is reported elsewhere[39] and is not specific to elephants. The presumption is, and experience supports, that there are no apparent unique or altered pharmacologic actions in elephants.

Desflurane, and most recently sevoflurane, are the two newest inhalation agents available commercially. Sevoflurane is qualitatively and quantitatively very much like isoflurane in many of its systemic actions in other large animals, such as horses.[36,40] Sevoflurane's main potential advantage over isoflurane is its low solubility in blood, which implies a shorter induction time, and more rapid recovery. Sevoflurane is presently expensive. It is also biodegraded (free fluoride ion is released) to a greater extent than the other contemporary inhalation anesthetics and is degraded in vitro in the

presence of $CO_2$ absorbents, such as soda lime and Baralyme, to an agent known as Compound A[11] that is known to cause renal injury in rats. Desflurane also exhibits a low blood solubility (even lower than sevoflurane). Its dose-related cardiovascular effects are no more depressing than those of isoflurane and sevoflurane and, at least at low doses, offer favorable advantage.[41,45] Desflurane resists biodegradation. On the other hand, it also is very expensive and requires a special, unusually expensive vaporizer for administration. Unlike other volatile anesthetics the desflurane vaporizer requires electricity to operate, limiting its portability. It is the least potent of the volatile anesthetics, and therefore inspired concentrations of $O_2$ will be reduced by 5–10% relative to the use of other agents. Although this should not pose a clinical problem in most circumstances, the possibility of this contributing to hypoxemia nevertheless exists in elephants.

**Alpha-2 agonists.** See "SECTION I: CHEMICAL RESTRAINT," for details on the use of these agents in elephants.

## Anatomy and Physiology

There are many important anatomic and physiologic considerations that may impact the anesthetic management of elephants. Although the ears of elephants are an organ of thermoregulation during anesthesia and recumbency, the efficiency of this function is markedly reduced. The ears are highly vascularized. The major arteries of the ear may be used to palpate and monitor pulse rate and quality. After the thick skin overlying an auricular artery is penetrated, it may be easily cannulated for measurement of arterial blood pressure and arterial blood sampling—for blood gas analysis, for example. The large auricular veins are the usual sites of choice for venipuncture facilitating venous blood sampling and perianesthetic balanced electrolyte/fluid administration.

The trunk of an elephant is important to anesthetic management in at least two ways. First, it is a prehensile appendage whose subtle movements are used as a sign of anesthetic adequacy. Second, elephants normally breathe largely through their trunks (70% or more of a breath[1]) so this organ must not be obstructed in an animal whose trachea has not been intubated. With regard to tracheal intubation it is important to consider that the oral-pharyngeal path to the larynx and trachea may be 80–100 cm in an adult, and passage between the dental arcade is surprisingly narrow for such a large animal.

Elephants should never be anesthetized in sternal recumbency. In the unconscious elephant, respiration is compromised because the abdominal viscera is compressed and exerts pressure on the diaphragm, inhibiting respiration.

**Normal function.** Normal values for various cardiopulmonary system–related tests are given in Table 9.7 and are useful for gauging anesthetic depth and physiological insult imposed by general anesthesia and recumbency. Data represent conditions in the same animals in the standing and the left laterally recumbent body postures; these elephants were not medicated. Cardiovascular values were similarly referenced to heart level.[17] Subsequently, other limited studies[19,30] add credibility to results highlighted in Table 9.7.

**Table 9.7.** Cardiopulmonary Parameters (Mean ± SD) in Unsedated African and Asian Elephants in Standing and Left Lateral Recumbent Positions[17]

| Parameters | Standing | Lateral Recumbency |
|---|---|---|
| Respiratory rate (/min) | 7.3 ± 0.2 | 7.5 ± 0.6 |
| Heart rate (/min) | 39.8 ± 0.8 | 46.3 ± 1.9 |
| Arterial blood pressure (mmHg): | | |
| Systolic | 179 ± 3 | 188 ± 10 |
| Diastolic | 119 ± 3 | 135 ± 7 |
| Mean | 145 ± 3 | 162 ± 7 |
| PCV (%) | 40 ± 1 | 41 ± 1 |
| Total protein (g/dl) | 79.2 ± 1.6 | 79.8 ± 3.4 |
| Hemoglobin (g/dl) | 11.1 ± 0.7 | 10.3 ± 0.4 |
| $P_aO_2$(mmHg) | 96.2 ± 1.6 | 90.3 ± 3.0 |
| $P_aCO_2$(mmHg) | 44.2 ± 0.5 | 43.3 ± 0.9 |
| $pH_a$ | 7.39 ± 0.01 | 7.41 ± 0.01 |
| Arterial base balance (mmol/L) | 1.7 ± 0.5 | 2.4 ± 0.7 |

## TECHNIQUES OF ANESTHETIC MANAGEMENT OF ELEPHANTS

### Preanesthetic Period

**Preparation.** Unlike the anesthetic management of most veterinary patients in a hospital environment, elephant anesthesia is usually an isolated event in a facility ill designed for this and for which people with various backgrounds and skills must be assembled. In addition, most equipment must be gathered from elsewhere and in timely fashion journey to the site (often unseen by key individuals prior to the event). As a result, general anesthesia for an elephant is not a trivial event and requires thoughtful consideration and extensive planning and preparation. Previous authors have highlighted the importance of detail in planning such an often intense, extensive, multifactorial event. Thoughtful planning must not be underestimated.[9,16]

Preanesthetic care. Feed and water are usually withheld from elephants for 36–48 and 12 hours, respectively. The elephant's prior physical condition, circumstances necessitating general anesthesia, and climatic conditions may modify this general recommendation. As with other herbivores, preanesthetic fasting of food and water is done to decrease bulk in the abdominal cavity and decrease the likelihood of intestinal bloat; either or both of these conditions increase the elephant's work of breathing or decrease its respiratory system perform-

ance and contribute to hypercapnia and/or hypoxemia. Special needs unique to the patient are addressed at this time as well.

Facility preparation and assembly of necessary equipment. Anesthetic and other appropriate support equipment is assembled and assured to be operational, including cuffed endotracheal tubes of appropriate size(s), associated intubation equipment, ropes to facilitate opening the mouth, a bite block to prevent closure of the mouth during manual tracheal intubation, a tracheal tube guide tube, and breathing circuit connectors. The anesthetic machine is readied, and components are verified functional and are leak tested. Special adaptations of the standard large-animal inhalation anesthetic machine (including breathing circuit) are often required to meet the needs of the larger tidal volume of an adult elephant. Such possible modifications include replacement of the typical, 30 liter large-animal rebreathing bag with three 30 liter bags arranged in series or replacement with a 150 liter weather balloon (Fig. 9.3). An alternative approach that has been used to meet the large tidal volume needs of an adult elephant is the use of two large-animal anesthetic machines joined in parallel at the breathing circuit Y-piece.[3] This arrangement has added advantages of additional fresh gas ($O_2$) inflow and anesthetic delivery capabilities and has been reported used in a variety of circumstances.[3,4,5,12,16]

Specialized monitoring equipment is prepared and may include equipment for electrocardiography; direct arterial blood pressure recording; pulse oximetry; and respiratory, anesthetic, and blood gas monitoring capabilities. Because monitoring equipment often requires electricity for operation, it is wise in preplanning to assure availability of sufficient electrical cords and electrical outlets in close proximity to the elephant recumbency site.

Provisions for prolonged recumbency should be prepared for. We have frequently used a 2.5 × 3.1 m waterbed especially prepared for elephant use (Fig. 9.4).[9,10] Alternatively, a thick bed of straw may be used. Special preparations for anticipated movement of the immobilized standing or recumbent animal need careful precise planning. This likely includes the need for a sling, hoist, or even a crane.[9,25]

**Preanesthetic medication.** Premedication is usually not necessary and in some cases not desired because the resulting sedative effect may make positioning the elephant for anesthetic induction difficult. In some cases mild, standing, sedation with a low dose of potent opioid or alpha$_2$ agonist administered intramuscularly may facilitate placement of a venous catheter or direct venous injection of the anesthetic induction drug following more precise animal placement. Atropine administration prior to anesthetic induction has been reported.[4,5,27] The author has not used it as part of his

***Figure 9.3.*** *A 150 liter weather balloon may be used as a rebreathing bag.*

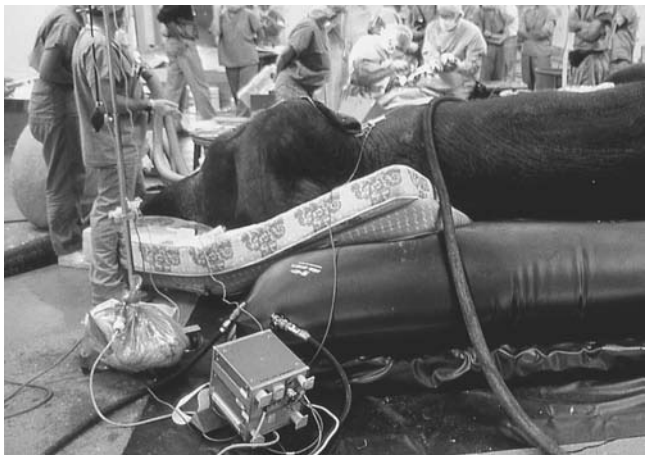

***Figure 9.4.*** *A specially prepared waterbed is recommended for prolonged procedures. See Chapter 10 for detailed description and another view.*

usual anesthetic plan but reserves its use (IV) when necessary in response to specific clinical circumstances, such as profound bradyarrhythmia during anesthesia. In experience to date, perianesthetic atropine use has not been necessary. Excitement in an elephant after IV administration of atropine has been reported.[12]

## Anesthetic Period

**Induction.** Induction is usually accomplished with the use of etorphine or carfentanil. Under the most ideal circumstances, a trained elephant can be positioned in lateral recumbency, an ear vein catheterized, IV fluid (balanced salt solution, such as lactated Ringers solution) administration started, and an induction dose of the potent opioid administered most safely by IV titration. On the other extreme is the untrained animal that is contained but uncooperative, perhaps even dangerous, and the opioid must be administered by dart or pole syringe in sufficient quantity to cause immobilization and recumbency. Obviously, one example is very atraumatic and the other potentially very traumatic both to the animal and/or facilities and attending personnel.

The uncooperative animal is also at heightened risk for drug overdose and attendant complications especially in inexperienced hands. Fortunately, the potent opioids in use have a relatively high margin for cardiovascular safety, and effective opioid agonist reversal agents are available; both of these minimize tragic outcomes.

**Tracheal intubation.** Following anesthetic induction, sterile ocular lubricant is applied and the animal is positioned as suitably as possible for tracheal intubation. The mouth is opened, usually with ropes, and a wood bite block placed between the molars of the upper and lower jaws on at least one side of the jaw to increase safety for the person performing the manual placement of the tracheal tube. The narrow dental arcades in elephants make tracheal intubation difficult. The routine used by the author is to palpate the oral and pharyngeal cavities manually to ensure a clear oropharyngeal passage through which to pass the endotracheal tube, If necessary, a large-animal–dose syringe or hose may be used to aid cleaning of the oral pharynx prior to intubation; the person administering the dose must be careful not to introduce water into the trachea.

After palpating the epiglottis and the two arytenoid cartilages, a guide tube (a 15–20 mm O.D. equine stomach tube) is passed through the rima glottis into the trachea. The intubator's arm is then withdrawn, and the free end of the guide tube placed through the tracheal tube (usually a 30–40 ID cuffed endotracheal tube for an adult elephant). The tracheal tube is then carefully manually guided between the dental arcades (with care not to lacerate the endotracheal tube cuff) and subsequently into the trachea, using the stomach tube as a guide.

**Maintenance of general anesthesia.** Following orotracheal intubation, the tracheal tube is connected to the Y-piece of the previously $O_2$-primed inhalation anesthetic machine. Fresh $O_2$ inflow is initially set at 10–15 l/min to facilitate denitrogenation of the circuit. When it is desirable to initiate delivery of inhalation anesthetic, the fresh gas flow to the breathing circuit is reduced to no more than 10 l/min to reduce inaccuracies between the inhalation anesthetic vaporizer dial setting and actual vaporizer output (i.e., at high flows, the vaporizer most commonly delivers less than dialed concentrations, especially at dialed anesthetic concentrations greater than 2%).[37] Oxygen flows during usual circumstances of anesthetic maintenance are commonly set at 5–10 l/min.

The technique of anesthetic maintenance is commonly one of two types: primarily inhalation anesthetic agent based (injectable agent [opioid] supplemented) or primarily injectable agent (opioid) based (inhalation agent supplemented). The primarily inhalation (or high dose inhalation agent) based technique is analogous to the technique most commonly used with other veterinary patients. Injectable drugs are used to induce anesthesia, followed by a transition to inhalation anesthesia with the background of injectable drug levels decreasing with time of anesthesia as a result of biodegradation and excretion.

Anesthesia is manipulated by altering the delivered dose (the vaporizer setting) of inhalation anesthetic. However, when using this technique the anesthetist must be mindful of the very large internal gas volume of the inhalation anesthetic delivery apparatus in use compared to that used for smaller species. Such circumstances markedly reduce the speed of change in inspired anesthetic concentration, which translates into perhaps an unexpected slowed increase or decrease in anesthetic level.[35]

With the primarily injectable agent (or low inhalation anesthetic dose) based technique, following intubation and a brief (15 min) inhalation anesthetic circuit priming period, the vaporizer is set at a relatively low anesthetic concentration (1–2% isoflurane). This provides low nearly constant background inspired levels of inhalation agent throughout much of the course of anesthesia, while swings in depth of anesthesia are manipulated by time and intermittent bolus injections of a potent opioid based on signs of anesthetic lightening. The maintenance technique selected depends largely on prevailing conditions (i.e., individualized technique). The author prefers the primarily injectable (opioid) based technique because it has provided suitable anesthesia without marked cardiopulmonary depression. Such conditions have also minimized (almost to exclusion) the need for use of vasoactive or cardiac inotropic support drugs and ventilatory assist devices. We value this because both drugs and added mechanical devices add complexity to preparation for, and conduct of, anesthesia.

With either technique an appropriate-sized inhalation anesthetic machine is used. This offers advantage in providing an increased inspired $O_2$ concentration relative to just breathing ambient air and reduces the likelihood of hypoxemia. Depending on the duration of anesthesia and the type of anesthetic machinery used, consideration should include the real possibility that the machine's $CO_2$ absorbent may have to be changed at some point during anesthetic management. The absorbent (e.g., soda lime, Baralyme) volume of a large-animal inhalation anesthetic machine is typically not great enough to provide suitable $CO_2$ absorption for prolonged (more than 2–3 hours) anesthesia.

**Monitoring.** Monitoring during general anesthesia is performed to enhance the quality of health care delivered. It provides data that reflects adequacy of anesthesia and patient safety. Palpation of a peripheral pulse (an auricular artery) to monitor rate and pulse quality, counting respiratory frequency and observing for behavioral signs of light anesthesia, is usually adequate for short-term chemical restraint and recumbency, but not for prolonged general anesthesia. Knowledge, skill, and technology for monitoring patients during general anesthesia have dramatically improved during the past two decades and have saved countless lives, including elephants. Equipment to monitor vital organ function has broadened in scope, has improved in accuracy under varying conditions, and has become more portable. Reductions in price have made improved monitoring more affordable and therefore accessible. To not monitor prolonged anesthesia is unacceptable.

Table 9.8 provides an overview of a cardiopulmonary monitoring scheme for general anesthesia.

Monitoring cardiovascular performance. Any of the standard bipolar limb leads (I, II, III) or the augmented unipolar limb leads (aVR, aVL, aVF) or the base-apex bipolar lead may be used for an ECG. See Chapter 24.

Arterial blood pressure should be monitored directly (via catheter positioned percutaneously in an auricular artery) or indirectly (measured at the tail via automated oscillatory recording from a sphygmomanometer, or Doppler technique). The direct technique is preferred because it is consistently most accurate, provides continuous information, and direct arterial access permits easy sampling of arterial blood for respiratory blood gas ($P_aO_2$, $P_aCO_2$) and acid base (pH) analyses.

Oximetry is a noninvasive device that for many years has been a standard of patient care for monitoring patients especially during general anesthesia. The equipment coordinates the measurement of arterial hemoglobin $O_2$ saturation ($S_aO_2$) with pulsations in arterial blood vessels. The equipment thus provides a reading for both $S_aO_2$ and pulse rate. An anesthesia monitoring protocol that includes pulsoximetry has also been described for free-ranging African elephants.[28]

**Table 9.8.** A Monitoring Plan for Elephants During Prolonged General Anesthesia and Recumbency

1. Eye changes associated with general anesthesia
2. Muscle tone
   a. Trunk tone
3. Response to stimulus
4. Preoperative hematocrit (packed cell volume) and total solids (plasma protein) concentration
5. Cardiovascular system
   a. Mucous membrane color
   b. Capillary refill time
   c. Heart rate and rhythm via electrocardiogram
   d. Palpable arterial pulse rate and character
   e. Direct arterial blood pressure
6. Respiratory system
   a. Frequency of breathing
   b. Depth of breathing
   c. Character of breathing
   d. Pulse oximetry
   e. Capnography (end-tidal $CO_2$ concentration)
   f. Inspired $O_2$ concentration
   g. Arterial (and/or venous) $PO_2$, $PCO_2$, pH

There is no standard for placement of the pulse oximetry sensor probe. The edge of the ear is usually convenient, but the probe is not always functional at this location. Sometimes cleansing the ear site and perhaps scraping the epidermal debris permits an accurate reading. If this does not work, other sites are tested, such as locations of the lip, tongue, or skin edges of the labia or prepuce. An $S_aO_2$ greater than 95% is always desirable.

The capnograph provides a continuous display of the $CO_2$ concentration (or partial pressure; the capnogram) in the inspired and/or expired breath. The $CO_2$ concentration measured at the end of exhalation (end-tidal or $ETCO_2$) provides a close estimate of the alveolar $CO_2$ concentration or alveolar $PCO_2$ ($P_ACO_2$). When the distribution of inspired gas in the lungs (ventilation) and lung blood flow (perfusion) are well matched the $P_ACO_2$ closely approximates the $P_aCO_2$ as measured by the $CO_2$ electrode of the blood gas machine. Both pulse oximetry and end-tidal $CO_2$ monitoring experiences have been reported.[26]

Precise information regarding respiratory gas exchange is obtained by measuring the $P_aO_2$ and $P_aCO_2$ (expressed in units of pressure; mmHg or kPa). Arterial blood is the preferred source because the sample mirrors events in the lung, not the peripheral tissue blood flow ($P_vO_2$ and $P_vCO_2$). Electrodes specific to the measurement are mounted in a device known as a *blood gas machine*. Measurements include pH. Highly portable devices are now also commercially available at affordable prices for measuring blood gases with accuracy nearing a standard laboratory bench-mounted blood gas machine.

Monitoring level of anesthesia. Depth of general anesthesia is typically defined by observing and categorizing progressive loss of function. Observations of various body

**Table 9.9.** Guidelines for Judging Depth of General Anesthesia in Elephants

| Sign of Anesthesia | Light | Deep |
|---|---|---|
| Heart rate | Generally rising* | Generally decreased* |
| Heart rhythm | May vary* | Regular* |
| Arterial blood pressure+ | Increased | Decreased |
| Breathing rate | Generally rising* | Generally decreased* |
| Ventilatory volumes | Generally rising* | Generally decreased* |
| Character of breathing | May vary* | Usually regular* |
| $ETCO_2$ or $P_aCO_2$+ | Normal or near normal | High (>55) |
| Eyelid aperture+ | Eyelids closed | Eyelids open |
| Eye position | Not central | Central |
| Pupil size | Small* | Large* |
| Palpebral reflex | Brisk | Slow or none |
| Lacrimation | Present | Absent |
| Trunk movement**,+ | Present** | Absent |
| Limb or tail tone/movement+ | Present | Absent |
| Shivering or trembling+ | Present | Absent |
| Swallowing+ | Present | Absent |

*Values diverging from normal; magnitude of change variable and/or drug dependent.
**Usually precedes limb movement.
+Among the more consistently reliable signs.

systems and functions include heart rate and rhythm (ECG, and/or pulse oximeter), arterial blood pressure, respiratory rate, depth and character of breathing (observe the rebreathing bag movement), end-tidal $CO_2$ or $P_aCO_2$, openness of the eyelids, position and/or movement of the eye, pupil size (though often confounded because of opioid drug use [miosis]), palpebral reflex, lacrimation, trunk tone, presence of gross limb or tail movement, shivering or trembling, and presence or absence of swallowing. Some broad, general indications of light or deep general anesthesia are given in Table 9.9. No one sign of anesthesia is infallible, and all are circumstance dependent. Judging depth of general anesthesia remains part of the art of the practice of clinical anesthesia.

**Mode of ventilation.** With carefully controlled anesthetic management, spontaneously ventilating elephants are usually normocapnic or only mild hypercapnic ($P_aCO_2$ of 50–60 mmHg), based on personal experience and supported by reports of others using comparable techniques.[3,4,5] Facilities for mechanical ventilation are usually not necessary, which markedly simplifies both preparation and anesthetic management. On the other hand, although sedation of standing elephants with contemporary drugs or recumbency in unsedated individuals does not usually cause hypoxemia,[14,17] heavy sedation or general anesthesia and accompanying recumbency usually at least reduces $P_aO_2$ to levels less than normal ($P_aO_2$ = 80–100 mmHg) or results in hypoxemia in air-breathing elephants at sea level.[4,5,43] Conditions are expected to worsen as elevation from sea level increases. Some $O_2$ enrichment of inspired gas is usually necessary. The most desirable and effective means is via tracheal intubation and breathing from an inhalation anesthetic machine. However, this may not always be possible or desirable and various other strategies have been reported, such as experience with insufflation of $O_2$ via one or both nostrils[6] or the endotracheal tube when available.[14] Successful improvement in $P_aO_2$ via insufflation is very much dependent on the terminal positioning of the delivery catheter within the respiratory tree. For most success the catheter must be positioned at least into the trachea and the closer to the carina the better. Of course high $O_2$ flow rates (e.g., 15–20 l/min) are also necessary. Another report described passing a small cuffed endotracheal tube through one (plugging the free nonintubated nares) or both external nares and allowing the elephant to breathe from a standard large-animal anesthetic machine from these tubes.[44]

Finally, use and effectiveness of one or two demand valves[18] have been reported. Although each of these techniques enrich the inspired air with $O_2$, none of them is suitable for use during anesthetic management plans employing inhalation anesthetics. However, they will provide varying benefit for supporting arterial oxygenation during brief periods of recumbent sedation with injectable drugs, an early phase of anesthetic recovery, or resuscitative efforts.

Although fabrication is possible, the authors are unaware of any mechanical ventilators of sufficient design to routinely ventilate adult elephants adequately. For brief periods, intermittent positive pressure ventilation has been delivered to anesthetized elephants using ventilators designed specifically for use in anesthetic management of horses.

**Hemodynamic support.** In the author's experience direct hemodynamic support is rarely necessary. The animals are usually systemically healthy and respond ap-

propriately to lightening of anesthesia. On the few occasions in which it was judged necessary, additional measures were warranted usually for only brief periods while anesthetic level adjustments were being made, using techniques similar to those used in horses. This included IV titration of blood pressure with the aid of an inotrope such as dobutamine. Because broad experience (personal and reported) with inotrope or vasopressor use in elephants is limited, the anesthetist must be vigilant for the development of life-threatening cardiac dysrhythmias.

### Immediate Postanesthetic Period

The paramount goal for successful recovery from general anesthesia is to have a calm, alert, atraumatic transition from anesthesia and recumbency to a standing posture. Fortunately, as a general rule, elephants tend to recover from anesthesia more like cattle rather than displaying the more fractious nature of horses under similar circumstances.

The technique used to facilitate a desirable recovery depends heavily on the technique and duration of anesthesia. In general, as procedures are nearing completion, the animal is detached from instruments in a systematic way, but retaining venous access. The amount of delivered inhalation anesthetic is also systematically reduced. The timing and speed with which anesthetic decrease is performed varies with circumstances. However, an important principle to keep in mind is that because of delivery equipment and animal size, inhalation anesthetic washout from both the animal and the breathing circuit will occur more slowly with elephants compared to similar circumstances with smaller species.[35,38] The expected rank order of speed of recovery (fastest to slowest) is desflurane > sevoflurane > isoflurane > halothane. The overriding goal is to minimize the likelihood of residual inhalation anesthetic contributing to ataxia and possibility of trauma in the attempt to rise to a standing posture.

When appropriate, the opioid component of the anesthetic technique is antagonized with an available opioid antagonist.[3,42] Depending on agents used (both agonist and antagonist) and time and dose of administrations, possibility for patient renarcotization (high dose of long acting versus short acting and/or low dose of antagonist) within a few hours of anesthetic recovery should be considered.

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# 10 Surgery and Surgical Conditions

Murray E. Fowler

## INTRODUCTION

Surgery for correction of certain disorders is an important methodology in the armamentarium of a veterinarian. Both domestic and wild animals are benefited by surgical intervention if an appropriate diagnosis warrants it. A number of challenges face the veterinarian contemplating surgery on an elephant, particularly if the animal is an adult.

Before electing to perform an elephant surgery, veterinarians and elephant managers should ask themselves the following questions:

1. Will the benefits of the surgery justify the risk to the elephant?
2. Will elephant care staff be at a special risk preceding, during and after the surgery?
3. Can the elephant be positioned to provide access to the surgical site?
4. Is a surgeon with elephant expertise available to perform the surgery?
5. Are facilities for anesthesia (equipment, agents, and experienced personnel) available?
6. Is there reasonable assurance that the elephant will be able to rise following the anesthesia and surgery?
7. Are emergency drugs and monitoring instruments available?

## GENERAL PRINCIPLES OF ELEPHANT SURGERY

### Presurgical Considerations

**Environment for the surgery.** Juvenile elephants up to 1000 kg may be immobilized and transported to a proper surgical theater. Surgery for them is similar to that performed on livestock and wild animals. Because adult elephants are so large, it is usually not possible to perform surgery in a surgical suite. A few zoos have now designed hospitals with doors of sufficient height to allow elephants to enter an enclosed surgery. Circus elephants and other highly trained elephants managed in free contact may be taken to an indoor facility and laid down for anesthesia.

The vast majority of elephant surgeries are performed in the enclosures where they live. Sometimes the elephant house may be enclosed for a degree of climate control; however, none of the facilities seen by the author would be considered to be an optimal site for aseptic surgery. It is little wonder that wound infection and dehiscence is a common sequel to major elephant surgery.

What steps should be taken to decrease the microbial fauna in an elephant building? First, air movement should be reduced. Air conditioning and fans should not be used. If time permits, the room should be hosed down with a high pressure water system and detergent washing. This should include the ceiling, walls, and flooring. After the detergent has been washed off, the structures should be sprayed with a disinfectant solution (sodium hypochlorite [bleach] or sodium-O-phenylphenol). The disinfectant solution must remain on the surface for at least 30 minutes and then rinsed off thoroughly with clean water.

If abdominal surgery must be performed outside, the institution should rent a large tent to erect over the immobilized elephant, or if the elephant is maintained in free contact it may be walked into the tent, but only if the elephant is able to lie down on command. The tent will minimize air movement and dust contamination of the surgery. Insect control should be carried out to prevent fly contamination of the incision and instruments.

All personnel who will be in the immediate vicinity of the elephant should be dressed in surgical attire, including face masks and gowns.

**Padding.** Prolonged recumbency of a large elephant places significant pressure on bony protuberances of the head and tuber coxa and the brachial plexus. Efforts should be made to pad these areas. If the elephant is trained to lie down on command, a waterbed is the ideal

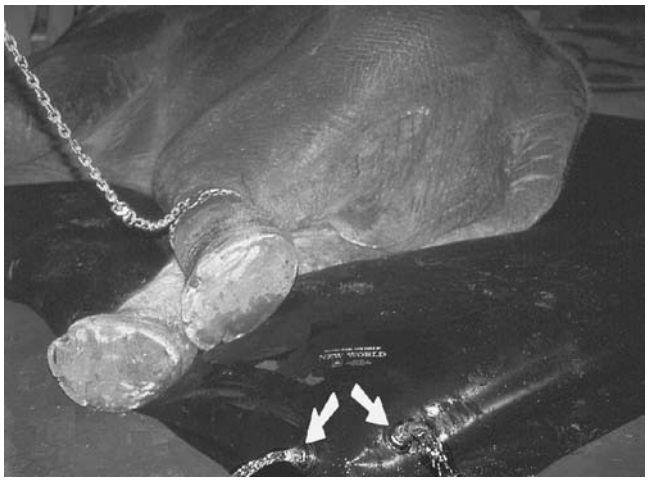


***Figure 10.1.*** *A waterbed for elephant surgery showing hose ports for filling and draining.*

padding.[20] A custom-made water bed has been successfully used on numerous occasions[22,23] (Fig. 10.1). (New World Manufacturing Inc., 27627 Dutcher Creek Road, Cloverdale, California 95425). The bed is 3.36 by 4.27 m (12 by 14 ft) and 38 cm (15 in) thick when fully inflated. The bed was constructed of 30 mm polyvinyl geotextile fabric, with four hose ports installed on one end. Only two of the ports are used to fill the bed, but all four ports are opened to drain the bed. The bed fills with the standard pressure of a household culinary system even under the weight of an adult elephant.

The empty water bed is situated and the elephant directed to stand on it. (This may require prior training, accomplished by asking the elephant to walk onto a black plastic sheet). The elephant is then directed to lie down. After the elephant is in position, the waterbed is filled.

Deep straw bedding may be used as an alternative to a waterbed. When immobilizing intractable elephants, plastic covered foam pads or sacks stuffed with straw may be thrown under the hip or head as the animal assumes recumbency.

**Instrumentation.** Standard large-animal surgical instruments are suitable for elephants. Instruments should be sterilized in an autoclave. An incision may easily be accomplished with a number 4 replaceable blade scalpel handle and a size 20 blade.

In a hospital setting, it is usual for all of the instruments to be in a single tray. When performing abdominal surgery in an elephant it is better to have smaller sets of instruments. One for the initial skin incision, another for opening the muscle layers, and still another to close the incision. Extra sterile blades and instruments should be available in case contamination or droppage should occur. Cold sterilization in a disinfectant solution is not suitable.

Instruments for special procedures will be discussed with the surgical technique. Laparoscopy is now being used in elephants.[68]

**Sutures and needles.** Most of the suture materials available currently have been used to close the abdominal wall of elephants. The suture pattern and placement are the keys for the strength of the incision closure, not the size of the suture material, which should be the same as for equine surgery.

Number 2 braided polygalactin (Vicryl®, Ethicon, Somerville, New Jersey 08876, USA) may be used to close the peritoneum.[16] Either number 2 or 3 Vicryl are suitable for other buried sutures. Skin closure should be with a monofilament fiber (polydioxanone [PDS, Ethicon], polypropylene [Prolene®, Ethicon] or [Vetafil®, S. Jackson, Alexandria, Virginia 22303, USA]). A monofilament fiber is less likely to wick infection from the skin surface to deeper tissues. Metal staples are not suitable for elephant skin closure.

Standard equine sutures with curved, swaged needles are appropriate. If these needles are used to close the skin, it is wise to sterilize needle-nosed vise-grip pliers to provide the strength necessary to penetrate the skin.[16] Foerner also recommends a sharp Gerlack suture needle (Western Ranch Supply, 303 N. 13th St., Billings, Montana 59103, USA, wrs@wtp.nwt) or a large curved postmortem needle to close the skin.

**Hemorrhage control.** Although elephants have a large blood volume (8% of their body weight—a 3000 kg elephant has approximately 240 liters [63 gal] of blood), hemorrhage control is necessary to prevent excessive blood loss and to allow visualization of the surgical field.

Hemostasis of the distal limb is a challenge because the skin is thick and inflexible and makes effective application of a tourniquet difficult. An inflatable cuff, as used in equine surgery, is ideal. Otherwise, it may be necessary to encircle the limb proximal to the carpus with a 12.7–19.0 mm (0.5–0.75 in) rope loop with sufficient slack to insert a 0.5 m (1.5 ft) hardwood stick (broom or shovel handle) to twist the rope loop and apply pressure. Another rope loop should surround the limb more proximally to anchor the end of the stick and maintain the twist. The author suggests that nylon rope not be used because it stretches too much. Additional twists may be applied to increase pressure on vessels during surgery.

**Withholding feed and water.** Withholding feed is a standard practice for elective surgery involving immobilization and anesthesia. Reasons for presurgical fasting include avoidance of regurgitation and reduction of the bulk of ingesta in the gastrointestinal tract. This reduction allows exploration of the abdominal cavity in order to locate testicles or obstructive lesions in the intestinal

tract and decreases gastrointestinal fermentation and potential tympany. Decreasing the volume of ingesta also decreases diaphragmatic pressure in a recumbent elephant.

Disadvantages of fasting include decreased energy available for metabolism at a time when energy demands are high, potential for altered metabolic processes, and increased stress associated with decreased nutrient intake.

Recommendations for fasting range from 24–72 hours in the literature. The author recommends 24–48 hours. Justification for 72 hours could be made only for castration of an adult bull. Juvenile elephants should not be fasted for more than 36 hours.

Water should not be withheld for more than 12 hours.

### Anesthesia

Optimal anesthesia is the most crucial factor for a successful surgery. Often elephant managers and administration officials expend considerable funds to bring in an experienced surgeon, but they neglect to employ an experienced anesthesiologist. This is unwise, because both may be crucial for success. Anesthesia will not be discussed in the sections on individual surgical procedures. See the section on anesthesia for details.

**Managing pain.** Elephants experience pain similarly to domestic livestock and horses. Current research demonstrates that postsurgical pain management is important for rapid recuperation from surgery. See Chapter 15 for details.

## POSTSURGICAL CARE

Surgery is not successful until the elephant has fully recovered, including incision healing and return to normal activity. Adequate postsurgical care is crucial to the success of many surgeries. Usually such care is performed by elephant handlers under the supervision of the staff veterinarian or elephant manager. Written instructions should be provided to the handler.

The possibility for optimal postsurgical care depends on the management system used and how well the elephant is trained to allow bandage changes and repeated medication. The care may be accomplished under both free and protective contact systems with proper training. One can't rely on an elephant restraint device (ERD) to force an elephant to accept treatment. The elephant will quickly refuse to enter the ERD.

### Indications

Repeated medications, particularly those that should be administered IM or IV, irritate the most docile and well-trained elephant. Select medications with prolonged activity to minimize the number of injections that must be administered.

Wound dressings may need to be changed. This is particularly necessary for foot surgery. Special sandals may need to be constructed to dissuade an elephant from removing the bandage. Arrangements for a 24-hour watch may be necessary.

Abscesses usually require lavage to maintain patency of the draining tract. Elephants are notorious for stuffing debris into open cavities such as abscesses and dental surgery.

Elephants often rub incision sites and vigorous rubbing may loosen sutures and traumatize the incision. It was necessary to repeat an amputation of the tail because the elephant rubbed the incision repeatedly until all the sutures were removed. A buried suture pattern was used the second time so sutures were not exposed to rubbing.

## SELECTED SURGICAL DISORDERS

### Abdominal Surgery

**Indications for celiotomy.** A number of cesarean sections have been performed in elephants, with poor results.[49] Prolonged labor devitalizes tissues plus sepsis and toxemia make the female a poor surgical risk. Numerous castrations have been performed and several attempts have been made to correct abdominal hernias.[1,34,50,57,61] Surgery of the gastrointestinal system has not been reported because antemortem definitive diagnosis of gastrointestinal problems is extremely difficult.

**Anatomy.** Any successful surgery requires a knowledge of the anatomy to be encountered during the surgery. Unfortunately there is no textbook of general elephant anatomy. Miall, in 1878, provided a text on the anatomy of the Asian elephant.[42] Mariappa provides a treatise on the dissection of a late-term fetus.[39] The latter two books are out of print but may be found in North American libraries. Many libraries provide interlibrary loan services so it is possible to see copies of these books. Grussen reviewed the literature of both Asian and African elephant anatomy.[27]

Abdominal surgery presents the most challenge to elephant surgeons. The following describes abdominal wall anatomy. See Figure 10.2.

The skin has variable thickness, 2.5–3.8 cm (1.0–1.5 in). The most superficial muscle of the abdomen is the cutaneous trunci muscle. Dorsally its aponeurosis blends with the thoracolumbar fascia. Muscle fibers are directed obliquely ventrad and craniad, becoming more horizontal ventrally, as they insert on the medial aspect of the elbow. Muscle fibers from both sides meet at the ventral midline. Contraction of this muscle produces skin twitching that discourages insects from remaining on the skin.

Beneath the cutaneous trunci muscle lies the abdominal tunic, which is a thick sheet of fibroelastic tissue. This is the suspensory support for the tremendous

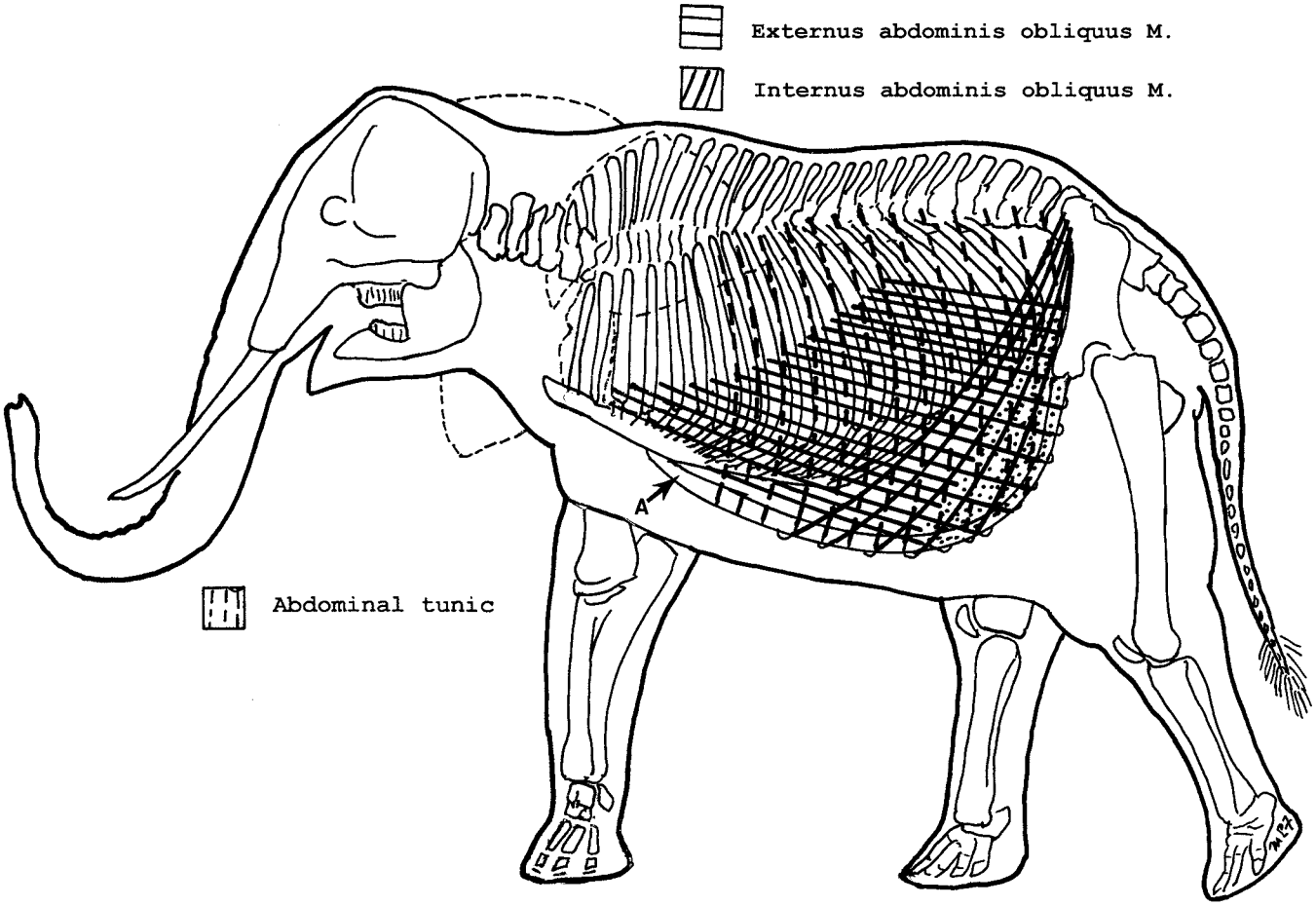



***Figure 10.2.*** *Diagram of the abdominal wall layers of an elephant. A) rectus abdominis muscle.*

weight of the abdominal viscera. The abdominal tunic is closely adhered to the superficial aspect of the external abdominal oblique muscle. Surgeons must identify this layer and incorporate it into any suture pattern used to close an abdominal incision.

The external abdominal oblique muscle is a thick, fleshy muscle with fibers directed caudad and ventrad from the costal arch to the iliac crest. At the midline the aponeurosis interdigitates with the fibrous sheath surrounding the rectus abdominis muscle. The external abdominal oblique muscle is also a major support for the abdominal wall. Every effort should be made to incise through this muscle in the direction of the fibers.

The internal abdominal oblique muscle originates at the iliac crest. Its fibers are directed craniad and ventrad, as in other mammals, to insert on the ribs and blend with the aponeurosis of the external oblique muscle to form the outer wall of the fascial sheath of the rectus abdominis muscle.

The paired rectus abdominis muscles lie on the floor of the abdomen and do not approximate at the ventral midline. Each muscle has its own fascial sheath. Fibers arise from the sternum and costal cartilages and are directed caudad to insert on the pubis of the pelvis via the prepubic tendon.

The transverse abdominal muscle originates as an aponeurosis that blends into the lumbodorsal fascia, the iliac crest, and the costal arch. The ventral aponeurosis inserts deep to the rectus muscle and forms the internal wall of the rectus sheath.

Deep to the muscles the surgeon encounters a thick retroperitoneal fat layer and then the extremely thickened peritoneum. Surgeons who may be accustomed to being able to penetrate the peritoneum with a finger will find that the elephant peritoneum must be grasped and incised with a scalpel or scissors.

**Presurgical preparation.** See the discussion under general principles in this chapter.

**Surgical technique.** The selection of an incision site is dependent on the abdominal organ to be accessed. A ventral midline abdominal incision should be avoided because there is no muscle support and only one layer of support fascia available for suturing. If a ventral abdominal approach is considered necessary, make a longitudi-

nal incision through the belly of the rectus abdominis, which has a heavy fibrous sheath providing two fascial support layers and the rectus muscle for suturing.

The author's preference for a celiotomy incision is to incise the skin on the lateral abdominal wall ventral and caudal to the costal arch, and with a slight curve dorsally. This allows exposure of the cutaneous trunci muscle that may be transected. The tunica abdominalis is closely adhered to the external abdominal oblique muscle, which should be incised in the direction of the muscle fibers. Deeper muscles may be separated or transected depending on the amount of exposure that is necessary.

Surgical technique for dealing with an abdominal organ is the same as for a horse.

**Incision closure.** Preparation for an abdominal incision closure should include redraping the incision site to prevent dragging suture material over possibly contaminated drapes. The surgeon should reglove and use a new surgical pack for the closure. Meticulous attention to incision closure is a key to a successful celiotomy.

Lateral wall closure. Either of the following closures have been used successfully. Each abdominal wall layer may be sutured separately, or the author's recommended closure is as follows (see Fig. 10.3):

Suture the peritoneal layer with a continuous suture pattern (Fig. 10.3). The transversus abdominal, internal abdominal oblique, and external abdominal oblique muscles and the abdominal tunic are closed together in a figure-8 tension suture (Fig. 10.3). Tighten sufficiently to bring layers into apposition, but don't compromise vascularity with a tourniquet effect. If edges of the abdominal tunic are not in apposition after placement of the tension suture, place simple interrupted sutures to close the gap. The cutaneous trunci muscle is closed with a simple interrupted pattern. The skin is sutured using a modified vertical mattress suture pattern (Fig. 10.3).

Paramedian incision closure. Suture the peritoneal layer with a simple continuous pattern. The fascia deep to the rectus abdominus, the rectus muscle, the fascia superficial to the rectus, and the abdominal tunic may be closed with a vertical mattress pattern (Fig. 10.4) or a figure-8 tension suture pattern. In either case, if the edges of the fascia are not apposed, place interrupted sutures to close the gap.

**Causes of incision dehiscence.** Incision dehiscence is a common sequel to abdominal surgery. Listing some of the causes should direct the surgical team to carry out procedures that minimize dehiscence. Dehiscence may be caused by infection, improper closure of the surgical incision, sutures breaking, knots slipping, tissue trauma during surgery, and seeping of serum or hemorrhage.

Infection is a major cause of wound dehiscence. Elephant skin has numerous creases or crevices that make it extremely difficult to obtain surgical asepsis during the scrubbing phase of presurgical preparation. Elephant

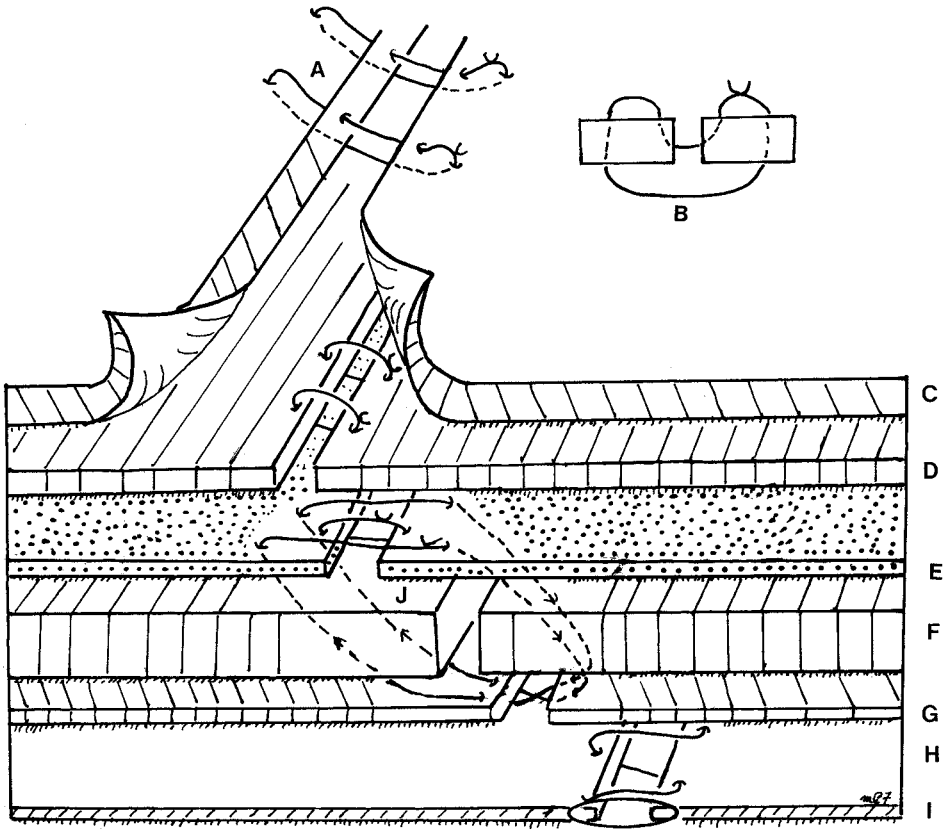



***Figure 10.3.*** *Diagram of the closure of an abdominal incision. A) modified vertical mattress suture, B) cross section of a vertical mattress suture to illustrate placement, C) skin, D) cutaneus trunci muscle, E) abdominal tunic, F) external abdominal oblique muscle, G) internal abdominal oblique and transverse abdominal muscles, H) retroperitoneal fat layer, I) peritoneum, J) cruciate tension suture.*

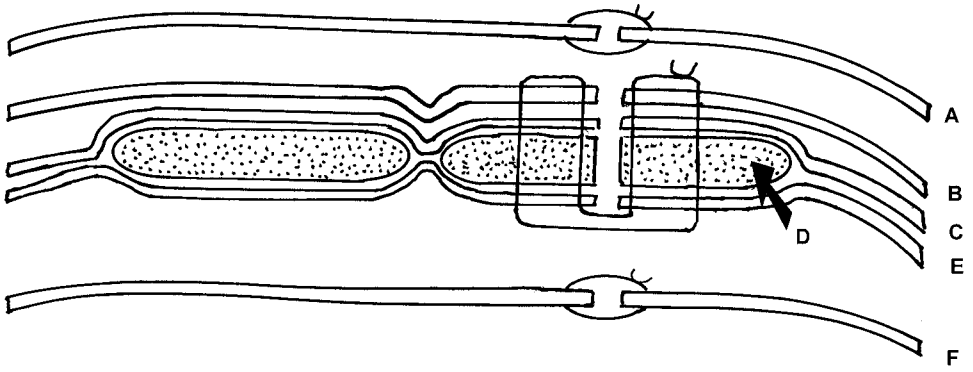



***Figure 10.4.*** *Diagram of the closure of a paramedian incision. A) skin, B) abdominal tunic, C) aponeurosis of the external abdominal oblique M, D) rectus abdominis M, E) aponeurosis of the transversus abdominis M, F) Peritoneum.*

***Figure 10.5.*** *Abdominal surgery being performed in the open.*

surgery is often performed in surroundings that are anything but ideal (Fig. 10.5). Contamination of the incision site from dust may occur. Gloves are easily contaminated. Drapes may become soaked with blood and wick infectious agents from areas that were not included in the surgical preparation. Sutures may be draped over areas that are not sterile.

## Castration

**Indications for castration.** Indications for castration of elephants are similar to those for domestic animals, such as control of aggressive behavior. This is important with elephants because the intact male exhibits a periodic behavioral change called *musth*. During musth, males become aggressive and extremely dangerous unless there are special facilities to control them. Castration abolishes the development of musth and the unpleasant behaviors associated with it, if castration is performed before any of the aggressive behaviors commence. See Chapter 32 for more details on musth. Aggressive behavior may develop in either male or female elephants at any time whether the male is castrated or not.

In Asia, male elephants were selected for work in the timber industries. Males were larger and stronger and they had tusks that aided them in their work. When the bull came into musth it was tethered in isolation to avoid injury to people. Unfortunately, handlers sometimes did not recognize the early signs of musth and may have been killed by what appeared to be sudden aggression.

Similarly, in the early 20th century bull elephants were popular for performing and exhibition because they were large and had tusks. When males matured and started to exhibit musth it resulted in some of the males being euthanized.

Although castration of an elephant is thought to be of recent origin, it was carried out as early as 1900.[59] The first castration in North America was performed in California by the author.[18,19] Other castrations have been performed in Germany,[24] Zimbabwe,[14] and the United States.[8,9,16,46,47]

Chemical castration using concentrated lactic acid has been attempted, but with unsatisfactory results.[16]

**Anatomy.** The testicles are intraabdominal and situated slightly caudal and medial to the caudal pole of each kidney. The stock is composed of mesorchium, testicular vessels and nerves, the ductus deferens, and fat, all surrounded by peritoneum (visceral tunic).

The stock was 5 cm (2 in) in diameter in a 9-year-old male. The chains of two equine écraseurs were broken while attempting to cut the stock.[18] Each testicle is elliptical in shape and approximately 13 by 20 cm (5 by 8 in) in diameter. Size varies with the age of the elephant. The surface of the testicle is smooth and is firm to palpation. Fecal balls in the colon are approximately the same size, but have a softer consistency.

**Presurgical preparation.** Fasting for 48–72 hours is important to minimize the volume of colonic ingesta.

**Anesthesia.** General anesthesia is required. Both injectable and inhalation anesthesia have been used. See the section on anesthesia for details.

**Positioning.** Successful removal of the testicle has been performed while in either right or left lateral recumbency. In elephants <5 years of age, both testicles have been reached from a left lateral position. An equine écraseur was sufficient to crush and cut the stock. In older animals it may be necessary to excise one gonad, close that incision, and wait 3–4 weeks before entering from the opposite side.

**Surgical technique.**

Incision. Several different approaches to the abdomen have been utilized. Access to the testicle is limited because of the narrow space between the last rib and the tuber coxa and the near-vertical positioning of the pelvis. The author has entered the abdomen through the intercostal space between the last two ribs, but prefers to remove the last rib.

In this approach, a skin incision [25–35 cm (10–14 in)] is made over the last rib near its origin. Discard the skin incision scalpel and use a new scalpel to incise muscle and fascial layers down to the bone. The superficial periosteum is incised and reflected back with periosteal elevators. The rib is transected dorsally with an oscillating bone saw or using a sterile obstetrical wire that is threaded around the rib between the bone and the periosteum. The periosteum is then dissected away from the deep aspect of the rib from dorsal to ventral and the rib removed. The periosteum provides a solid layer for wound closure.

An alternative approach is to stretch the hindlimb caudally with a block and tackle or a trucker's hitch (Fig. 10.6). This procedure opens the upper-flank area maximally for optimal exposure. The incision is made midway between the last rib and the tuber coxae.[16]

In either approach, the retroperitoneal fat layer may be as much as 30 cm (12 in) thick and is difficult to deal with. The author removes fat, controlling hemorrhage with ligation, until the peritoneum can be grasped with a Vulcellum forceps. It is not possible to poke a hole through the peritoneum with a finger as is done in equine surgery. The peritoneum is lifted into the incision and cut with a scissor.

An alternative approach to peritoneal penetration is to grasp the testicle through the peritoneum and then incise the peritoneum over the testicle with a scalpel.[18,19] After the peritoneum is incised, enlarge the opening manually to allow insertion of a hand. Grasp a margin of the peritoneum with a long forceps to maintain a landmark for future insertion of an arm or instrument.

Within the abdominal cavity, search for the kidney and locate the testicular stock caudal and medial to the kidney.

Removal of the testicle. The method for amputation of the testicle depends on the age of the elephant and the diameter of the stock. In animals <5 years of age, an equine écraseur may be used to crush and cut the stock. The écraseur chain is carried into the abdomen over the arm of the surgeon. When the testicle is identified and grasped, the chain is opened and slipped over the testicle and pushed proximally as far as possible. Keep the chain snug to prevent any other viscus from being caught in the chain. The chain is tightened to crush the vessels and left in that position for 15 minutes before completing the cutting. Maintain a grasp on the testicle when making the final cut.

Other methods for transecting the stock are necessary in animals over 5 years of age. Self-locking stainless steel bands have been used.[16] The author had a modified equine écraseur manufactured using a motorcycle chain for added strength.[19] This instrument was used to crush the stock and vessels as described above. Then a fetotomy apparatus was used to place an obstetrical wire loop distal to the écraseur to cut the stock.

A third technique was initially to transect the stock using obstetrical wire. The spermatic artery could be

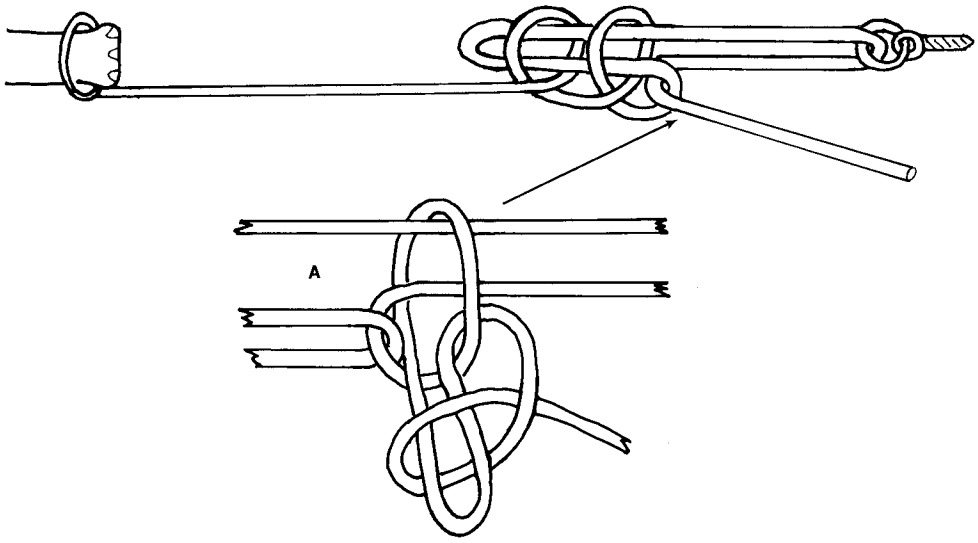



***Figure 10.6.*** *Schematic diagram of a trucker's hitch that may be used as a block and tackle to anchor an elephant limb to a ring on a wall or to a post. A) Finishing hitches to fix pressure that has been applied.*

palpated digitally in the remaining stump and could be grasped with a large-curved Kelly forceps.[16] The isolated spermatic artery could then be crushed with an equine écraseur.

Incision closure. This closure is the same as for general abdominal surgery.

## Osteitis of the Phalanges

Foot infection is a disorder in elephants that is dealt with in Chapter 20. Surgical treatment to drain abscesses and remove foreign bodies may help prevent osteitis.[3,11,21]

**Indications for surgery.** Osteitis may be a sequel to the spread of infection from behind the toenail, foreign body penetration of the sole, or a subsole abscess near the periphery of the foot. Osteitis may be limited to phalanx-3 (P-3), but if it is not treated effectively, the infection may spread to other phalanges and adjacent tissues.

Some success in medical management has been obtained by regional digital perfusion with appropriate antibiotics, but more commonly surgical removal of infected bone is the only viable option.

**Diagnosis.** Usually a wound is being treated and there is a fistulous tract evident behind a toenail or in the adjacent sole. A radiograph provides the definitive diagnosis, but a fistulagram may further characterize the extent of the tract leading to the bone. See SECTION II of Chapter 13.

**Presurgical preparation.** Presurgical antibiotic therapy should be administered at least 24 hours prior to surgery, based on culture and sensitivity of material aspirated from the fistulous tract following insertion of a stiff-sterile catheter into the tract. Early in the presurgical preparation, the wound and fistulous tract should be lavaged to diminish the volume of exudate present during surgery. Then the fistulous tract should be packed with gauze soaked in a disinfectant.

The surgical site will be contaminated with numerous microorganisms, but every effort should be made to minimize spread. Intense cleaning and disinfection of the entire foot prior to anesthesia is required. The toenails, cuticles, and soles should be trimmed at an earlier time to minimize preanesthesia stress. If possible the forward surface of the toenail should be rasped to near the corium in preparation for an incision through the nail to obtain maximum exposure of the infected bone(s).

Arrange for an appropriate tourniquet. A sturdy table should be constructed with a 38–50 cm (15–20 in) square surface and legs 60–76 cm (24–30 in) long. This table is used to straddle the down limb and support the upper limb. This support relieves some of the pressure on axillary or inguinal vessels and nerves and provides better access for the surgeons.

**Positioning.** The elephant should be positioned for maximum exposure of the affected toenail and digit. After the elephant is recumbent and anesthetized, the limb should be elevated and rested on a padded table. The table should be covered with a sheet of plastic to minimize contact of drapes with the wood.

**Presurgical cleaning and disinfection.** Finish rasping the forward aspect of the toenail down to the corium. The entire foot should be cleaned with a recently disinfected stiff-bristled brush, followed by standard surgical aseptic preparation of the site. The final application of the disinfectant should be allowed to dry before drapes are applied. The limb above and below the surgical area should be wrapped with a nonpermeable drape, including the bottom of the foot. After the surgical scrub is completed, the site should be covered with a sterile towel until surgical drapes are applied.

**Anatomy.** The third phalanx, if present, is behind the toenail. The distal end of P-2 extends below the upper toenail. P-1 is proximal to P-2. Digital vessels and nerves are located on either side of the digit. Digital extensor tendons are located on the superficial aspect of the phalanges.

**Surgical technique.** The vertical incision is made through the center of the toenail and is extended dorsally 20 cm (8 in).[13,20,22,23] Deeper tissues are incised to the bone in the lower area to expose the infected bone. Retraction for exposure is accomplished by large animal rake retractors held by assistants. Hemorrhage control is obtained by tightening the tourniquet. Do not attempt to ligate vessels.

Infected bone segments are removed with large animal Cloward rongeurs or curettes. Intraoperative radiographs insure removal of all infected bone segments. P-2 may be disarticulated from P-1 for removal in toto. If P-1 appears to be infected, it may be transected above any infected bone using an oscillating surgical bone saw.

When all infected tissues have been removed, a final lavage with povidone iodine is done. The skin of the incision is closed with a vertical mattress pattern. The fistulous tract and external wound should be debrided, lavaged, and packed with surgical gauze soaked in povidone iodine solution. The lower end of the incision is left open for drainage, and then the foot is bandaged.

**Postsurgical care.** Failure of the wound to heal may be caused by spread of infection into adjacent tendon sheathes or failure to remove all infected tissue. Postsurgical wound care may be prolonged (months) and necessitate changes based on cultures and sensitivity during the process.

## Dental Surgery

**Indications for surgery.** The most common dental problem is failure of the cheek teeth to extrude laminae as the teeth migrate rostrally. Fractures of a tusk are also common. Management of tusk fracture may be as simple as smoothing the edges of remaining segments; if the pulp cavity is exposed, pulpitis may result in the need to extract the tusk root.

**Anatomy.** Review the discussion on dental anatomy described in Chapter 22. During the rostral migration and wear of each cheek tooth, the rostral laminae are shed. The tusks of male Asian elephants and both male and female African elephants are modified incisors that continue to grow throughout life. The distal extent of the pulp cavity is approximately 15 cm (6 in) beyond the emergence of the tusk from the sulcus of the skin.

**Signs of failure to extrude laminae.** The elephant may be reluctant to eat. The exposed roots of the retained laminae cause gingivitis. An oral examination reveals the segments extending rostral to the dental alveolus. A foul odor may emanate from the mouth.

**Surgical technique for removal of laminae.** General anesthesia is required. Metal chisels and a mallet are required to produce a groove between the segment to be removed and the normal tooth. The mouth may be held open with a wooden block wedged between the upper and lower arcades. The wedge shape of the chisel is usually sufficient to break the laminae off. In one case a custom-made chisel was constructed from an iron bar 2.5 cm (1.0 in) in diameter and 45 cm (18 in) long. The chisel was struck with a 7.25 kg (16 lb) sledge hammer, but the laminae didn't chip off even with repeated attempts. Ultimately the tooth was found to be loose in the alveolus and the entire tooth segment was extracted by hand. No postsurgical care is necessary.

**Signs of tusk pulpitis.** A discharge may issue from the tip of the open tusk at the fracture site. A necrotic bone odor may be present. The tusk root may be movable in the alveolus. The base of the tusk is near the maxillary sinus, and pulp infection may extend to the bone and into the sinus.

A normal reaction to a fracture involving the pulp cavity is for the pulp to begin forming dentine to seal the tusk against pulpitis. Failure to form a dentine layer allows infection to progress proximally, and it may ultimately involve the entire pulp cavity. A dark spot at the tip of a blunted tusk is the result of restorative dentin formation, which is brown to dark brown. It is an indication of pulp exposure and potential chronic pulpitis.

An elephant tusk may be fractured during intraspecific fighting, by a fall into a moat, or during immobilization. Chronic pulpitis may cause the pulp to shrink away from the tusk wall, which may result in thinning of the wall of the tusk root, eventually causing weakness and possibly an additional fracture. An elephant may also break a tusk purposefully by trying to pry or thrust at objects in an enclosure. In Asian range countries, a tusk may fracture when tusks are used to move heavy logs in timber operations.

**Surgical techniques for dealing with tusk fractures.** Many reports are in the literature on managing tusk fractures.[2,5,7,37,52,63,64,65,67] Consultation with a dentist experienced in endodontic procedures is recommended before embarking on an elephant dental procedure. Fractures may occur anywhere from near the tip to the root at the level of the orifice of the dental alveolus. Fractures occurring distal to the pulp cavity present no medical problem other than the necessity of smoothing off sharp edges. The fracture line is usually at a diagonal, which minimizes the potential for the fracture line extending proximally to involve the pulp cavity.

A recent fracture (1–2 days) of the tusk involving the pulp cavity should be managed as for a vital pulpotomy. Sufficient pulp should be removed to allow a layer of calcium hydroxide paste to be placed against the pulp and a second layer of dental restorative to form a solid plug in the pulp canal (Fig. 10.7).

Hemorrhage may be controlled by applying an epinephrine-impregnated sponge to the pulp surface. Soak a cotton pledget with 1% formacresol solution to disinfect the pulp surface. The tusk wall is prepared to hold a dental restorative plug by drilling a bevel or groove around the internal wall with a hobby drill burr (Dremel Moto-tool, Dremel, P.O. Box 1468, Racine, Wisconsin 53401, USA). After the calcium hydroxide layer is in place, the tusk cavity is dried and a dental restorative

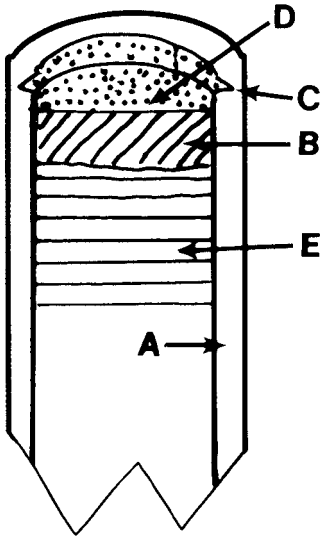
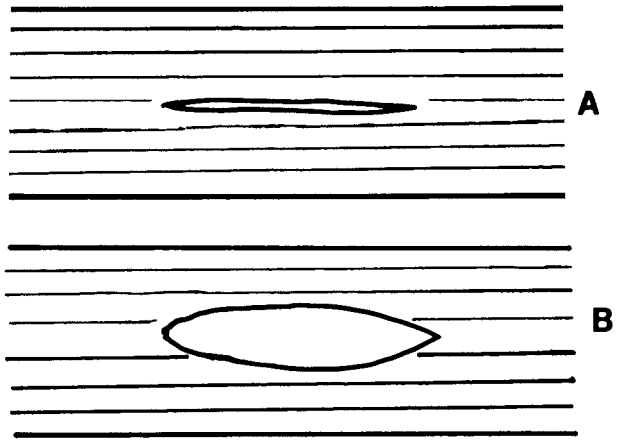



***Figure 10.7.*** *Diagram of a longitudinal section of a fractured tusk using a partial pulpectomy for correction. A) Tusk wall, B) zinc oxide layer, C) wall beveled to anchor, D) the dental restorative plug, E) pulp.*



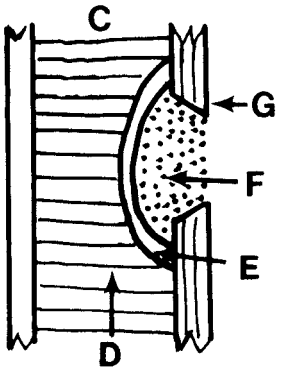




***Figure 10.8.*** *Diagram of the management of a recent longitudinal fracture of a tusk using a partial pulpectomy. A) Original fracture line, B) tusk wall sculpted to allow access for partial pulpectomy, C) cross section to illustrate placement of layers, D) pulp, E) zinc oxide, F) dental restorative plug, G) beveled wall to anchor plug.*

(glass ionomer or amalgam) plug pressed into place. The plug should be flush with the orifice of the tusk.

Longitudinal fractures of the tusk are difficult to manage; however, vital pulpotomy may be performed on a recent fracture (<2 days) (Fig. 10.8). Metal rings are sometimes applied to a longitudinal fracture on the theory that the metal ring will prevent further extension of the fracture line. The author has reservations about the effectiveness of metal bands.

#### Tusk extraction.

Indications for tusk extraction. Continued intermittent exudation that is refractory to medical management is the most common indication. Looseness of the tusk in the alveolus indicates a loss of the periodontal membrane and ultimately loss of the tusk. An alveolitis may occur that causes a stronger fibrous attachment and even alveolar bone proliferation, making it more difficult to extract the tusk.

Surgical techniques for extraction. A number of techniques have been described.[2,5,7,37,51,64,66] Each may have application in selected cases. The reader is directed to the original papers for details. The following description illustrates an effective procedure.[66]

If the fracture is distal to the skin sulcus, the tip of the tusk should be sawed off to expose the pulp cavity. If proximal to the sulcus, dorsal and ventral skin incisions are made to allow reflection of the skin flaps to reveal the margin of the alveolus. A variable speed surgical drill or osteotome burr is used to remove dentine from the pulp cavity to reach pulp tissue. Any remaining pulp tissue should be removed by using stainless steel hooks and curettes mounted on long handles. The opening should be enlarged to allow insertion of a narrow-bladed wood saw.[66]

The tusk wall is sectioned longitudinally with four or five cuts. A thin-narrow chisel must be custom manufactured to separate the tusk wall from the alveolar bone by cutting the peridontal membrane. Traction on the wall segment is maintained with an equine dental forceps or a pair of pliers.

When all the segments have been extracted, the alveolus should be scraped using a long-handled curette. Hemorrhage should be minimal after the segments are removed. The alveolus is packed with a sterile 8 cm (3 in) gauze bandage soaked in povidone iodine solution. It is important that the packing be in one unit to avoid leaving packing material in the alveolus when changing the packing or lavaging the alveolus (three times per week for 3–6 weeks). Skin incisions should be closed with vertical mattress sutures.

Postsurgical care. Antimicrobials are routinely administered for 10 days. Trimethoprim sulfa has been found to be effective.[66]

### Miscellaneous Surgical Procedures

Numerous other surgical procedures have been performed on elephants, including mammectomy,[62] tumor extirpation,[4,26,30,43,51,65] amputation of the tail,[17] kidney biopsy,[31] trunk lacerations,[10] fetotomy,[28,29] prolapsed nictitating membrane,[32] vestibulotomy for dystocia and artificial insemination,[40,54,55,56] gunshot wound care,[58] vaginal prolapse,[12] esophagotomy,[60] and fracture repair.[48] Papers describing general surgery in elephants include George,[25] and Kuntze.[33,35] Equine surgical techniques are satisfactory for dealing with these basic surgical procedures.

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# 11 Infectious Diseases

Murray E. Fowler

## INTRODUCTION

### Host Relationships

The fundamentals of microorganisms and infectious disease require an understanding of hosts.[74] A *reservoir host* is one that harbors a microorganism and serves as a source of infection for other species. In order to show that a particular species is a true reservoir host, the following four criteria must be met. First, the reservoir host must be able to maintain the microorganism in the absence of other species. Second, at some time the host must shed the microorganism to allow infection of other species. Third, transfer of the microorganism from the wild host to another species must be demonstrated. Fourth, the reservoir host is usually not seriously affected by the microorganism, though some individuals may show overt signs.[74]

A *definitive* or *primary host* is the species in which a microorganism passes the adult and/or sexual stage of the life cycle. A definitive host may also be a reservoir host. An *intermediate host* is one in which the microorganism passes the larval or nonsexual phase of the life cycle. An *amplifier host* is one in which the microorganism pool is built up, without necessarily causing severe overt disease. The larger pool is then available to spread to a more susceptible host. An *aberrant host* is an unnatural host for a microorganism that usually suffers severe overt disease. A *vector* is a mechanical means of spreading a microorganism from one animal to another.[74]

### Nidus Concept

Ample evidence shows that infectious agents and parasites coevolved with specific groups of animals. As the virulence of the microorganism increased, the host adapted in some manner so that the population would not be exterminated by the disease. It is to the advantage of the microorganism, as well as that of the affected host, that any disease produced be relatively mild to enable both to exist perpetually.

Infectious agents in a stable ecosystem have a nest home or habitat which is called a *nidus*.[216] The natural microorganism nidus exists under definite conditions of climate, vegetation, soil, and favorable microclimates in those localities in which vectors, donors, intermediate hosts, and recipients of the microorganism occur. Thus the nidus of a microorganism is characteristic for a definite geographical landscape. A stable infectious agent relationship also necessitates suitable conditions within or on the host. The nidus concept is a somewhat simplistic explanation of host/parasite relationships for long-term coevolution. However, infectious disease is a dynamic process, and some microorganisms are capable of rapid adaptation to new hosts and new habitats. Numerous factors are involved in host specificity, including ecologic, physiologic, and anatomic considerations, but of most importance is the evasion of the host's immune response through the mounting of an effective immune suppression response, providing antigenic disguise or modulating surface antigens.[74]

It may be difficult to impossible to identify the nidus of many microbial species because the pristine balance has been upset, primarily because of human interference. Domestic animals have been moved all over the world. Wild animals (potential reservoirs) also have been introduced into new areas. Vectors and intermediate hosts have been imported or exported with their hosts. Vegetation has changed with agricultural practices and the lumber industry. Wetlands have been drained and developed into industrial tracts or homes. In zoological collections, animals from different geographical regions that would not be close in the free-living state are often housed in close proximity and sometimes commingled. Movement of wild animals between zoological gardens may be a factor.

Some microorganisms infect a broad spectrum of animals. Others are almost species specific. For some microorganisms, a true reservoir host is unknown.

**Table 11.1.** Laboratory Test Terms and Abbreviations

AGID = Agar gel immunodiffusion
AGP = Agar gel precipitin
BC = Bacterial culture
CF = Complement fixation
CPE = Cytopathic effect on tissue culture cells
ECE = Embryonate chicken eggs
ELISA = Enzyme-linked immunosorbent assay
EM = Electron microscopy
DNA = DNA amplification (specific DNA primers)
HA = Hemagglutination
HI = Hemagglutination inhibition
IFA = Immunofluorescence antibody test
IHC = Immunohistochemistry
MAP = Monoclonal antibody panel
NI = Neuroaminidase inhibition
PCR = Polymerase chain reaction
RIA = Radioimmunoassay
RT = Reverse transcriptase
SVN = Serum virus neutralization
TC = Tissue culture
VI = Virus isolation
WIBA = Western immunoblot assay

## Stress

From a clinician's standpoint, immune suppression is of paramount importance. Stress is a major external factor that interferes with immune competence. Excessive and prolonged stress has neurohormonal effects on most organ systems of the body. Of particular importance in the context of immunosuppression is hyperproduction of cortisol (Fowler 1986).

## Laboratory Diagnostic Procedures

*Laboratory diagnostic procedures* are referred to throughout this chapter. Abbreviations for these tests are listed in Table 11.1. For an explanation of these tests, see Hirsh.[119]

# IMPORTANT VIRAL DISEASES

## Elephant Endotheliotropic Herpesvirus (EEHV) Infection

**Definition.** Herpesvirus infections are found in most mammalian species. Primary hosts are rarely severely affected by the virus, but secondary hosts such as Asian elephants are. Herpesvirus infection in elephants is characterized by generalized hemorrhages in the heart, liver, intestine, and tongue.[31,29,30]

**Etiology.** Herpesvirus inclusions were first observed in pulmonary nodules in asymptomatic elephants.[9,29,30,66,70,102,128,174,173] Herpesviruses were not isolated then, but novel endotheliotrophic herpesviruses were identified by DNA technology from elephants that were severely affected in the 1990s in the United States.[240] Two herpesviruses may be found in elephants. The first herpesvirus was noted during a culling program in Kruger National Park in South Africa, where workers found many elephants with nodules in their lungs. These nodules revealed intranuclear inclusion bodies consistent with herpesvirus infection. All the elephants were asymptomatic. The second herpesvirus was noted in skin papillomas and vulvar lymphoid lesions. Both EEHVs occur in the African elephant as the primary (reservoir) host, one of which affects Asian elephants that develop acute and often fatal disease. The second herpesvirus has been associated with fatalities in two captive African elephants in the U.S.[243]

**Epizootiology.** Little is known about the distribution and overall epizootiology of the nonlethal herpesviruses of the free-ranging African elephant**.** The initial outbreak in the United States began in 1995 and was followed by reports in 10 elephants in eight North American zoos (8 Asian and 2 African).[240,241] In Europe the index case was a circus elephant in Switzerland.[212,114] Three cases have been reported elsewhere in Europe in zoos[228] and one in Israel.[240] Between 1980 and 2005 there were 32 cases of EEHV in captive elephants worldwide, including those cases identified retrospectively (personal communication, Dr. Richard Montali, Davis, California, June 2005). Little is known as to how the virus is transmitted from animal to animal. The African elephant is the likely source of infection for Asian elephants, but Asian elephants may also transmit to Asian elephants.[243]

**Clinical signs in elephants.** The onset is sudden. Initial signs are lethargy, anorexia, and mild colic.[212] As the disease progresses, there may be edematous swelling of the head, neck, trunk, and thoracic limbs. A sign that is often seen is cyanosis of the tongue, which usually begins at the tip and progresses caudally.[242,244,239,243,240,241] Abortion has occurred with evidence of herpesvirus infection in fetal tissue.

**Diagnosis.** In three Asian elephants, hematologic findings included leukopenia, thrombocytopenia, and a low erythrocyte count.[240,241] In contrast, in an elephant that was treated successfully in Missouri, there was a leukocytosis (up to 20,000 per l) but with lymphopenia and a monocytosis.[259,260] Virus serum neutralization and immunoblotting have been used to identify infection antemortem.[240]

The virus may be detected early, during the viremic stage in blood, by polymerase chain reaction (PCR) technology. Although not suitable for screening blood from well elephants, PCR technology is also useful applied to tissue at necropsy (heart, liver, tongue, and intestines).[29] A serologic assay for glycogen-protein B (gB) shows promise for elephant blood.[72,29]

Gross lesions observed at necropsy include pericardial effusion with petechial and ecchymotic hemorrhages throughout the myocardium. The tongue is usually cyanotic. Hepatomegaly is generally present,[204] and

ulcers may be observed in the oral and laryngeal cavities and the intestines.[240,241]

Microscopic findings include microhemorrhages in the heart and tongue. Interstitial edema and some hepatocellular degeneration are observed in the liver. Basophilic intranuclear inclusion bodies may be seen by both light and electron microscopy in capillary or sinusoidal endothelial cells of affected organs.[240,241]

**Differential diagnosis.** Diseases causing sudden onset and rapid death—such as encephalomyocarditis, clostridial enterotoxemia, salmonellosis, toxicosis (sodium fluroacetate), and hypovitaminosis E—should be considered.[200,240]

**Management.** The prognosis for herpesvirus infection in elephants is unfavorable.[200] The peracute nature of this disease precludes treatment unless the condition is recognized early. Treatment has been attempted and successful in at least two elephants. In one calf, a diagnosis was made early and antiviral therapy begun with a 3-week course of famciclovir (Famvir, Smithkline and Beecham, Philadelphia) administered orally three times a day (TID).[258,259,260] The initial dose was 12 mg/kg TID, which was reduced to 4 mg/kg TID after allometric scaling was factored in. Broad-spectrum antimicrobial therapy was also administered to counter secondary infection.[259] A subadult bull was successfully treated in the Netherlands.[255] A pharmacokinetic study of famciclovir in Asian elephants has shown that 8–15 mg/kg administered orally or rectally should result in penciclovir (the active compound) concentrations considered therapeutic in humans.[125]

No vaccine is available for this herpesvirus. Since there is concern that the African elephant may be the source of infection for Asian elephants, it would be prudent to avoid housing African elephants with Asian elephants, particularly with young elephants.[251] Lack of facilities may make this difficult. Asian elephants may also be a source of exposure to other Asian elephants naive to earlier exposure, so risks of transmission of EEHV may be higher during elephant movements to new facilities. Serological tests to identify potential elephant carriers and previous exposure to EEHV are becoming available (personal communication, Dr. Richard Montali, Davis, California, June 2005).

## Encephalomyocarditis

**Definition.** Encephalomyocarditis (EMC) is a natural infection in rodents, without producing clinical disease. Sporadic outbreaks occur in a variety of domestic and wild animals, including elephants.[205]

**Etiology.** EMC virus is in the genus *Cardiovirus*, family Picornoviridae. The virus replicates in myocardial cells and kills them, causing the tissue damage that occurs.[290,285,310]

**Epizootiology.** Rodents are considered to be the reservoir host. EMC virus has been recovered from numerous species of wild rodents worldwide. Pigs are the primary domestic animal host, but many species of captive wild animals have been infected with a frequently fatal myocarditis. Both African and Asian elephants have developed EMC in South Africa, Australia, and the United States. Outbreaks of EMC in the United States have been primarily restricted to the southeastern states.[82,81,87,86,85,310]

Infection most likely occurs via the oral-fecal route from feed or water contaminated with rodent urine or feces. An outbreak of EMC in free-ranging African elephants in Kruger National Park in South Africa was correlated with a population explosion of multimammate mice *Mastomys natalensis,* which had a high prevalence of antibodies to EMC virus. The feeding behavior of elephants—i.e., pulling up and ingesting tufts of grass containing rodent nests—may have contributed to infection of the elephants.[103]

Cases in elephants have occurred sporadically in eight zoos in six states in the United States. Infection doesn't spread between elephants.

**Clinical signs in elephants.** Sudden death is the predominant sign or, in less acute cases, anorexia, lethargy, and moderate-to-severe dyspnea. These are signs associated with congestive heart failure. Myocarditis is the principle effect in elephants, resulting in pulmonary edema, hydropericardium, and ascites.[87,265,272,310]

Not all infections in elephants result in clinical disease. In a study in Kruger National Park, 53% of the animals had antibody titers to EMC virus.[103] There appears to be a sex bias toward clinical infection in males. It is known that testosterone enhances susceptibility to EMC infection.[78]

Recovery from infection may leave myocardial scars, which may cause problems later if an elephant is immobilized. The stress of the immobilization may place an additional burden on a weakened myocardium, making it susceptible to ventricular dysrhythmia caused by catecholamine release following administration of an immobilizing agent.[110]

**Diagnosis.** The infection progresses so rapidly that there may be little time to obtain samples for antemortem screening. Serologic tests may be used to survey other members of a herd.

Sudden death or signs associated with heart failure plus myocarditis at necropsy provide a presumptive diagnosis. A definitive diagnosis is made in acute cases by isolation of the EMC virus in tissue culture cells. Confirmation is by inhibition of infectivity or hemagglutination by antisera specific for EMC virus.[290] PCR techniques are also used.[278]

Gross lesions observed at necropsy include pale streaks in the myocardium, hydrothorax, and hydropericardium with fibrin in the fluids. Pulmonary edema

with froth in the tracheobronchial tree may be seen. This frothy trunk syndrome is seen associated with immobilization following recovery from EMC.

Microscopically, myocardial degeneration and necrosis predominate with lymphocytic infiltrates. Virus particles may be visible on electron microscopy.

**Differential diagnosis.** Any disease that causes myocardial necrosis must be considered, including endotheliotropic herpesvirus infection, hypovitaminosis E, and cardioactive glycoside plant poisoning (oleander *Nerium oleander*).

**Management.** There is no specific treatment for EMC. Prevention by rodent control is crucial. Rodent population monitoring would reveal a population buildup, enabling initiation of more rigorous control methods.

During an outbreak in Kruger National Park, elephants were vaccinated with an in-house–developed aziridine-inactivated vaccine in an oil adjuvant.[123] In clinical trials, vaccinated elephants withstood a potentially lethal challenge with EMC virus.[233] Inactivated vaccines were used in outbreaks in the United States with no conclusions reached as to effectiveness in elephants.[5,290]

## Foot and Mouth Disease

**Definition.** Foot and mouth disease (FMD, aftosa, aphthous fever, fiebra aftosa, fièvre apthouse, maul-und-klauenseuche, hoof and mouth disease) is a highly contagious but rarely fatal viral disease, primarily of cattle, sheep, swine, and goats, but also affecting other domestic and wild ruminants and nonruminants (armadillo *Dasypus novencinctus*, nutria *Myocastor coypus*, capybara *Hydrochaeris hydrocaeris*, elephant, and hedgehogs *Erinaceus europaeus*). FMD is characterized by vesicular lesions and, subsequently, erosions of the oral mucosa and skin of the feet.[74,290]

**Etiology.** FMD is caused by *Aphthovirus* sp. family Picornoviridae. Seven immunologically distinct types of FMD virus (FMDV) are known (A, O, C, Asia 1, SAT1, SAT 2, SAT3). Within the 7 types, over 60 subtypes have been identified by complement fixation tests.[7,17,111,112]

**Epizootiology.** The route of field transmission may be by respiratory aerosol, direct contact with infected animals, animate vectors (humans), inanimate vectors (vehicles, tools, instruments), or exposure to contaminated feed and water. FMDV can be inhaled in aerosol droplets by people working around infected animals. The virus lodges in the pharyngeal mucosa and may be exhaled for as long as 24 hours postexposure. Thus humans may contribute to the spread of FMD. Actual FMDV infection in humans is rare, and the disease is not considered to be a public health risk.

Free-ranging elephants are rarely affected when outbreaks occur in ruminants in their territory.[122,134] Thus, clinical FMD is a disease of captivity in elephants.[3,35,154,230,232,234,235,268,290]

**Clinical signs in livestock.** The incubation period varies from 3 to 5 days. In cattle, early signs include fever, depression, anorexia, and vesicle formation. Stomatitis causes salivation, and lameness is caused by lesions on the coronary bands and interdigital spaces. Pain may cause the animal to tread, shake or kick out the feet, or lie down. Ultimately, the vesicles rupture, forming erosions, and a mucopurulent nasal discharge may develop. Pregnant cows may abort and calves die acutely. The overall mortality rate in cattle is usually less than 5%, but 50% of affected calves may die from myocardial degeneration.

**Clinical signs in elephants.** Evans[69] described an outbreak occurring with a group of military elephants. The first signs noted were anorexia and lameness. Elephants had a mild fever. Vesicles were noted on oral examination on the tongue, palate, cheeks, and mucous membranes of the trunk, with an accompanying mucoid exudate from the nares. Examination of the feet revealed hot, swollen, and tender skin around the toenails and margins of the slipper. Lameness was severe in some cases, causing the elephant to remain recumbent. The incubation period was 3–4 days, with the illness lasting for 10–20 days in uncomplicated cases. The slipper may become undermined, resulting in sloughing.[154] In animals involving foot-slipper sloughs, nursing care may be required for months. Reluctance to eat results in a rapid weight loss. Coalescence of erosions may form ulcers that become scabbed over and fly larvae infested. Young animals may have a systemic response, with lesions in the myocardium and intestine causing diarrhea, dehydration, and sudden death. Two other authors report similar clinical signs in elephants.[232,235]

**Diagnosis.** Diagnosis of any suspected vesicular disease is under the control of state and federal regulatory veterinarians in the United States and comparable authorities in other countries. Any disease causing erosive and necrotic lesions of the nares and oral cavity should be considered. Suspected cases must be reported promptly. Suspect facilities must be quarantined until a diagnosis is confirmed or disproved by a variety of sophisticated laboratory tests. A provisional diagnosis will be made within 24 hours of receipt of the lesion material at the diagnostic laboratory at Plum Island, New York in the United States.

Any elephant with vesicular or erosive lesions of the oral cavity or trunk mucosa should be isolated from other animals until FMD has been ruled out. If an elephant can be handled, tags of vesicular epithelium should be placed in phosphate-buffered saline solution and kept refrigerated for virus isolation. Blood should be collected into CaEDTA.

Ten ml of serum should be obtained along with standard collection of fresh tissue and tissue in formalin. Definitive diagnosis is carried out only by government veterinarians in the United States, with the following tissues submitted to Plum Island: vesicular fluid, epithelium covering a vesicle, and five ml of blood in an anticoagulant. Rapid diagnosis may be done by EM, Immuno-EM, or PCR. Viral antigens may be identified directly from tissue via CF, AGID, IFA, and ELISA. The virus may be grown on tissue culture and/or by animal inoculation. Virus typing is performed by ELISA or PCR.[215,290]

Serologic tests are used in chronic cases and include SVN, ELISA, and a blocking ELISA.[134,290]

FMD is rarely fatal in adult elephants, but young elephants may develop mycocarditis and vesicular and erosive lesions in the intestines. FMD is the only vesicular disease occurring in elephants, but since the vesicular phase of FMD will likely be overlooked in elephants, any inflammatory lesions of the oral cavity or trunk might be included for consideration (herpesvirus infection, pox).

Strains of the FMD virus isolated from elephants in India include Type O, A, A22, and Asia 1.[17,234,7,215]

**Management.** With appropriate general nursing care, most elephants with FMD will survive and pose no threat to other animals. Provide soft feed to counter reluctance to eat. Foot-slipper sloughs will require prolonged dressings and the use of protective boots. There is no evidence to indicate that elephants remain a carrier following infection. Nonetheless, governments may dictate what should be done with an affected elephant. Vaccination is a complicated situation, with numerous antigenically distinct strains. Some countries, such as the United States and Canada, have a policy of euthanizing any affected animal, and the use of vaccines is prohibited. Public pressure on governments may prompt changes in regulations at any time, as experienced in the United Kingdom and France during a recent outbreak of FMD there. Vaccination may be recommended in some parts of the world in the face of an outbreak in livestock in the area.

## Elephant Pox

**Definition.** Elephant pox is a viral disease characterized by inflammation of localized areas of skin and/or mucous membrane and commonly resulting in generalized or systemic disease. The progression is first a papule and then a vesicle to a pustule and ulceration.[51,245,291]

**Etiology.** Originally, the virus identification of pox in elephants was linked to a vaccinia virus strain that was used for human small pox vaccination.[12,11,10] Elephant rides for children were common at that time in European zoos. It followed that the likely source of the infection in elephants was from children who had recently been vaccinated against small pox. Subsequent studies have shown that elephant pox virus is in the genus *Orthopoxvirus* sp. and is closely related to the cowpox virus.[139] It is a distinct strain, however, which may be differentiated by chick embryo lesions, plaque formation in cell culture, and intradermal tests in rabbits.[139,159,245]

**Epizootiology.** An outbreak of elephant pox was reported in India and Sri Lanka.[69] Between 1960 and 1986, 22 outbreaks occurred in elephants in European zoos.[138,141,129,213] Since elephant pox was an acute and sometimes fatal infection it was assumed that elephants were not the definitive host for the virus. Studies have shown that the elephant pox virus is closely related to a rodent pox virus.

Infection occurs by direct contact with virus-contaminated objects, other infected animals, contact with infected rodents (likely reservoir), or rodent predators such as the domestic cat.[226,227,224,225,222,223]

Elephant pox is a zoonosis, but close contact is generally required, such as an attendant who has close contact with an affected elephant.[170,168,169,247]

**Clinical signs in elephants.** Signs are variable, from mild conjunctivitis to systemic illness and death.[51,61,91,90,115,300,139] Conjunctivitis may become severe. An elephant may present with dysphagia, salivation, and difficult mastication as a result of pox lesions in the oral cavity and pharynx.[90] Pox lesions occur on the tongue, trunk, temples, abdomen, thorax, perineum, and vulva. The toenail and slipper corium may become infected, necrotic, and odoriferous.[90] The toenails of severely affected elephants may slough.[140,144,146,142,141,145,160,162]

Skin pox lesions are 1–2 cm in diameter and grayish yellow in color with a central hemorrhagic area. Lesions may coalesce, may become necrotic, and an area of the skin may slough. Lesions on the mucous membranes are primarily ulcerations. Skin pox lesions begin as a rash and progress to a papule, vesicle, pustule, and ulceration with scab formation. Elephant pox has caused abortion.[318]

**Diagnosis.** The hematologic picture is primarily a leukopenia. Eosinopenia may be absolute.[140,141] Screening is generally not applicable. Clinical signs are presumptive, but a definitive diagnosis is reached by histopathology, virus culture, and electron microscopy for Bollinger bodies.[159,186]

Grossly, the lesions appear as described in clinical signs. At necropsy, lesions may be observed on spleen and liver serosal surfaces, pericardium, and epicardium.[170,168] Internal ulceration of the mucous membranes of the pharynx, esophagus, and trachea may be seen. Eosinophilic intracytoplasmic inclusions are seen on light microscopy, and pox virus particles may be observed by electron microscopy.

**Differential diagnosis.** Papillomatosis is quite rare.

**Management.** General nursing care should include providing soft feed such as bran mash, alfalfa meal, and molasses and warmed water. Antibiotics may be appropriate to control secondary infection. Special nursing care may be required if one or more toenails or the slipper sloughs. Special boots and dressings are needed to protect the exposed corium until regrowth of the cornified tissue occurs (months). This has been successful in a few cases in Germany.

Adequate rodent control is essential. Vaccination has been employed in Germany with good success. Initially an MVA strain of vaccinia was used,[224] and later a Lister (Elstree) strain of vaccinia was used to halt an outbreak and prevent infection.[245]

## Rabies

**Definition.** Rabies is one of the oldest diseases known to man, being first described over 4000 years ago. It is characterized as a uniformly fatal encephalomyelitis except in reservoir hosts.

**Etiology.** The etiology is a *Lyssavirus*, serotype/genotype 1, in the family *Rhabdovirus.* The *Lyssaviruses* are thought to have evolved on the African continent, and in the present, variants of the virus are found on every inhabited continent.[249] A species of *Lyssavirus,* serotype/genotype 7, has produced clinical signs and encephalitis in Australian bats similar to rabies in bats elsewhere.

**Epizootiology.** Rabies has a worldwide distribution with the exception of Australia, the United Kingdom, Scandinavia, and a few smaller countries. Rabies is transmitted by bites from reservoir hosts or from any animal that becomes infected and exhibits signs. In a rabies context, reservoir refers to those species that are capable of maintaining the virus in nature, although many of those hosts will develop clinical rabies. Rabies virus is known to reside in reservoir hosts unique to geographical locations.

In the United States, the natural host is the spotted skunk *Spirogale putorius*. Aberrant hosts that may become infected and live long enough to be an effective transmitter of the virus include the striped skunk *Mephites mephites*, raccoon *Procyon lotor*, coyote *Canis latrans*, red fox *Vulpes vulpes*, gray fox *Urocyon cineroargenteus,* and various species of insectivorous bats (the silver-haired bat *Lasiomycteris noctiveagans* has caused rabies in human beings and llamas *Lama glama*). Each of these hosts may harbor a unique strain of the Lyssavirus that may be identified using monoclonal antibody panels. Forty years ago the domestic dog *Canis familiaris* was the primary aberrant host involved in the transmission of the rabies virus to almost any other species of mammal in the United States. Now, because of the vaccination programs carried out in dogs, wild animals are the principle reservoirs.

In South America the vampire bat *Desmodus rotundus* is the natural host, with numerous carnivores, human beings, and livestock being the aberrant hosts. The domestic dog is still the primary species responsible for transmission of rabies virus to other species.

In Africa, natural hosts include the civet *Civettictis civetta*, the zorilla (striped polecat) *Zorilla striatus* and a unique cycle is observed in which domestic dogs become infected, recover, and become carriers. Aberrant hosts include the African hunting dog *Lycon pictus*, various other species of carnivores and vivverids (mongoose).

In Asia and the Middle East, the dog is the predominant reservoir. Infection is spread to other canids, such as jackals, foxes *Vulpes vulpes arabica*, the raccoon dog *Nyctereutes procyonoides,* and some small mustelids.[249]

In Europe, the principle reservoirs are carnivores and insectivorous bats.

**Clinical signs.** There are no pathognomonic signs of rabies in any species. The time from the bite to initial signs may be as short as 3 weeks or as long as several months, particularly in wild animals.[3,13,69,93] The signs of rabies are similar in most species of mammals. Not every animal will exhibit all the signs, but when enough cases are observed the following signs may be noted. There are only a few cases of rabies in elephants; the general signs are listed so that clinicians may be cognizant of possible rabies.

Initial signs may be anorexia or behavioral changes, but these progress to neurological signs that may vary depending on the precise location of the encephalitis. In most species there is a neurologic excitement stage (hyperesthesia to visual, tactile, or auditory signals), sudden aggression (furious rabies), disorientation, pica, incoordination, and convulsions. Stimulation of the autonomic nervous system may result in hypertension, hyperventilation, muscle tremors, priapism, hypothermia, and/or hyperthermia.

The paralytic phase (dumb rabies) may follow the stimulation phase, or animals may become paralytic directly from the premonitory phase. Signs of the paralytic phase include lethargy, paresis, incontinence, tenesmus, tail flaccidity, ataxia, swaying, and asymmetrical paralysis and coma.[249]

The progress of a clinical case in an Asian elephant was described by Gupta.[106] An elephant was bitten on the trunk by a rabid dog in India. This elephant was examined by a veterinarian 2 days after the bite. He disinfected the wound with phenol and a week later began antirabies postexposure treatment with the Pasteur vaccine, 60 ml daily for 14 days. A month later it was seen by a veterinarian, who noted that the elephant was anorexic and had saliva pouring out of the mouth every 4 or 5 minutes.[106] Tears were flowing from both eyes. It was shivering and kept shifting weight on the hind legs. The body temperature was 37.4°C (99.3°F). The elephant was quiet and responded to the ankus. These signs had

begun 3 days previously. By the next afternoon, partial paralysis of the rear quarters was evident. The elephant began to vocalize. He fell three times, getting up each time, but was unable to rise after the fourth fall and died shortly thereafter.

In another case, an 84-year-old female Asian elephant was reported to be lethargic for 4 days. On the fifth day she was unsteady, aggressive, and restless with secretions from both temporal glands. By day 6 she was completely anorectic, had complete paralysis of the trunk, was unable to stand, and appeared to be blind. She died on the ninth day.

Antigenic typing determined that the virus strain was similar, but not identical to the local dog strain. There was no history of a dog bite in this case.[313]

**Diagnosis.** Rabies should be considered in any disease that has a central nervous system component. Caution should be exercised in examining and carrying out diagnostic procedures.

There are no infallible antemortem screening tests for rabies.[15] Although skin biopsy, corneal impression, and saliva collection for detection of the virus are used antemortem in some species, a negative finding doesn't rule out rabies.

The definitive diagnosis for rabies requires the histological examination of sections of the brain stem. Removal of the brain from the skull is no easy task, and it must be accomplished with safety for all concerned. Preferably, the person collecting the brain should have been preimmunized with a human rabies vaccine and have a demonstrated titer of 1:50 or more. He or she should be clothed in a washable long-sleeved garment, which is then covered with a plastic or rubber apron. The face should be masked and goggles worn to prevent flying particles from reaching the mouth, nostrils, or conjunctiva. Hands should be gloved or, better still, double gloved with one pair being heavy-duty latex. See Chapter 14 for details of removing the brain.

One half of the brain should be sent under refrigeration to the appropriate diagnostic laboratory in the county, state, or country. If this can't be done in a timely fashion, preserve the tissue in a 50% saline glycerin solution. The other half of the brain should be retained for routine histologic evaluation. Some diagnostic laboratories will refrigerate the saved half until the results of the rabies evaluations are reported.

All tools and equipment should be cleaned and disinfected immediately with a viricidal agent.

Encephalitis is not likely to be observed grossly at necropsy, but examination of the brain tissue for evidence of the virus is definitive.

The immunofluorescent antibody (IFA) test is now used globally as the standard diagnostic method. Impression smears are made from the brainstem, hippocampus, and cerebellum. The smears are fixed and fluorescin labeled monoclonal or polyclonal antirabies virus reagents are applied. Then the slides are examined by direct fluorescent microscopy.[249]

Caution must be used in applying the various techniques used in livestock to wild species such as the elephant. Even some domestic animals don't respond to certain tests. The dog, but not the horse, responds to the fluorescent antibody test. The author had cuts on his hands when he examined a horse that turned out to be rabid. The brain was submitted through appropriate channels and reported as being negative by IFA. The pathologist who conducted the necropsy and histopathology felt that the encephalitis present warranted further consideration. He injected brain tissue into mice and within 2 weeks they died with classic rabies. The antirabies Pasteur treatment was begun 2 weeks late!

Histopathology of standard tissue preparations of the same areas of the brain as mentioned before revealed a nonsuppurative encephalitis and Negri bodies (intracytoplasmic inclusion bodies). Caution again! Some species don't develop Negri bodies.[249]

The definitive diagnosis is done by virus isolation through animal inoculation or tissue culture, but RT/PCR may be used to confirm the presence of Lyssavirus nucleic acid. SVN antibodies in the CSF are also diagnostic because vaccination does not produce CSF antibodies. Serologic procedures for detecting rabies antibodies include SVN and ELISA.

When submitting the brain of an elephant or any wild animal to a laboratory for rabies diagnosis, it is prudent to request multiple tests to rule out nonresponse—e.g., IFA or Negri bodies.

**Differential diagnosis.** Differential diagnosis in elephants should include any disorder with a neurologic component, such as encephalomyocarditis, tetanus, botulism, trauma (gunshot wound, hit by a vehicle, skull fracture), or heavy metal poisoning.

**Management.** No treatment is available. Elephants suspected of having rabies or other neurological conditions should be isolated and handled only in a no-contact mode. Only inactivated (killed) rabies vaccine should be used in elephants or any other animal for which the vaccine has not been validated for efficacy and safety. No vaccine has been validated for use in elephants; nonetheless, clinicians are likely to vaccinate in endemic areas, such as the northeastern United States, where rabies in raccoons *Procyon lotor* has become epizootic.

In the United States, Imrab 3 TF (Merial, Duluth, Georgia) has been used on a wide variety of captive wild animals, including elephants. The National Zoo in Washington, D.C. experienced an explosion of some 27 rabid raccoons on zoo grounds. Over 1700 animals were vaccinated with the standard 1 ml dose of Imrab 3, including the elephants. All developed what are considered to be protective titers in other species. No challenge studies have been conducted to verify efficacy, but safety

is assured (personal communication, Dr. Lindsey Phillips, March 15, 2004).

## IMPORTANT BACTERIAL DISEASES

### Elephant Tuberculosis *(by Susan Mikota)*

**Definition.** Tuberculosis (TB) is an infectious bacterial disease affecting numerous species. TB occurs in primates, elephants, rhinoceroses, tapirs, domestic and nondomestic bovidae, other nondomestic ungulates, and psittacine birds.[199] TB has been known to occur in elephants for over 2000 years.[127] Reports from the 19th and early 20th centuries[25,6,50,84,207,286] described the disease in Asian elephants. The first report of TB in African elephants was in 1962.[95,96] Additional reports appeared in the middle of the 20th century.[100,107,133,229,254,267] In 1994 TB "emerged" as a disease of concern for captive elephants in North America, and between 1994 and 2005, 34 cases were confirmed (Mikota, unpublished). TB has also been reported in elephants in Europe.[152]

**Etiology.** Tuberculosis is caused by mycobacterium belonging to the *Mycobacterium tuberculosis*-complex (MTBC). Four species of tubercle bacilli—*M. tuberculosis*, *M. bovis*, *M. africanum*, and *M. microti*—comprise MTBC. Humans are the natural and reservoir hosts for *M. tuberculosis* and *M. africanum*. Domestic cattle are the natural and reservoir hosts for *M. bovis,* and *M. microti* infects the meadow vole. Among domestic and nondomestic animals, susceptibility to *M. tuberculosis* and *M. bovis* varies. Elephants are affected primarily by *M. tuberculosis,* although infection with *M. bovis* has occurred.

The terms atypical mycobacteria, nontuberculous mycobacteria, and mycobacteria other than tuberculosis (MOTT) refer to mycobacteria other than MTBC. The term *avian tuberculosis* refers to disease caused by *M. avium* complex (MAC; composed of *M. avium* and *M. intracellulare*). *M. avium* is commonly isolated from elephant trunk wash cultures but has not been associated with clinical disease.[191,192] A variety of nontuberculous mycobacteria has also been isolated.[217] For the most part, nontuberculous mycobacteria do not appear pathogenic for elephants. In two cases, however, an uncommon mycobacteria, *M. szulgai*, was associated with fatal disease (personal communication, Kathryn Gamble, DVM, Chicago, Illinois, March 2005). A novel, rapidly growing mycobacterium (*Mycobacterium elephantis*, sp. nov) was also isolated from a lung abscess in an elephant that died of chronic respiratory disease.[269]

**Epizootiology.** The primary mode of transmission of TB is by aerosolization of infected respiratory droplets. In humans, coughing is the most common aerosol source. Among elephants, aerosolization may occur during trunk spraying or during greeting when an infected elephant inserts its trunk into the mouth of a susceptible elephant. Aerosols may also be dispersed during necropsies and from tuberculous abscesses. Fecal-oral spread may be possible, but it has not been documented in elephants. *M. tuberculosis* has been isolated from the feces of infected elephants (probably related to organisms that were coughed up and swallowed).

Humans exposed to infected elephants have shown tuberculin skin test conversions.[187,209] In one reported case, an elephant handler and four elephants were infected with the same strain.[187] Tuberculosis has not been reported in free-ranging elephants.

TB can be transmitted only from elephants with active disease. Active disease may occur immediately following primary infection or (more likely) by reactivation of latent infection. Risk factors for transmission include bacterial load, length of exposure, proximity to the infected animal, immune status, droplet size, and ventilation.[163]

**Clinical signs in elephants.** Antemortem signs of TB are frequently absent. Chronic weight loss is the most common sign. Anorexia and weakness may occur and exercise intolerance may be seen in working elephants.[107,175,192,229,267] An abnormal discharge may be noted from the trunk. In one case, TB was diagnosed by isolation of *M. tuberculosis* from a vaginal discharge (personal communication, Dr. G. Dumonceaux, Tampa, Florida, June 2005).

**Diagnosis.** In the United States, Guidelines for the Control of Tuberculosis in Elephants have been developed and are administered by the U.S. Department of Agriculture (www.aphis.usda.gov/ac/ElephTBGuidelines2003.html). These guidelines specify procedures for diagnosis, treatment, and surveillance. As of this writing, identification of MTBC by culture is considered the definitive diagnostic test. Samples are obtained by a trunk wash procedure. The procedure is illustrated in Chapter 13. A culture series is recommended because bacteria may be shed intermittently. Any abnormal discharge should be cultured. The TB organism is slow-growing, and culture results may take up to 8 weeks. The intradermal tuberculin test, commonly used to diagnose TB in humans, cattle, and deer, shows poor sensitivity (16.7%) and specificity (74.2%) in elephants.[192] A multiple-antigen enzyme-linked immunosorbent assay has demonstrated 100% sensitivity and 100% specificity; however, limitations inherent in the study necessitate further research, which is currently underway.[149]

Two other new tests show promise for the diagnosis of TB in elephants. The Rapid Test (RT) is a screening immunoassay that uses a mixed grouping of selected mycobacterial antigens. Nitrocellulose membranes are impregnated with test antigens in a single test strip and placed into plastic cassettes similar to a pregnancy test kit. The multiprint immunoassay (MAPIA) is a confirmatory test that assesses the presence of antibodies to 10 individual mycobacterial antigens.[156]

Multiple serum samples collected from 90 Asian and African elephants in Europe, South Africa, and the U.S. have been tested using the MAPIA and RT. Of these, 17 were culture-positive for *M. tuberculosis* (or *M. bovis* in one African elephant). All 17 tested positive with MAPIA and/or RT (100% sensitivity). Of 63 culture-negative control elephants with finalized status (healthy or other disease), one was reactive with RT but not MAPIA (this animal had a chronic osteomyelitis). In all elephant TB cases where retrospective samples were available for testing, the antibody responses could be detected much earlier (2 to 6 years) than positive cultures from trunk washes were first documented. Further, when TB-infected elephants were under treatment, the antibody titers to certain antigens used in MAPIA declined quickly to baseline levels in response to therapy. Thus, MAPIA may prove useful not only for early diagnosis, but also for monitoring response to treatment (personal communication, Dr. K. Lyashchenko, Orlando, Florida, June 2005).

Tuberculosis may also be detected by nucleic acid amplification (Amplified *M. tuberculosis* Direct Test [MTD], Gen-Probe, San Diego, California, 92121, USA) performed on trunk wash samples. Results of this method correlate well with culture results.[49,192]

Also under investigation is an immunoblot assay. Using this method, antibody responses in TB-infected elephants to *Mycobacterium bovis* whole cell sonicate were detected 4 years prior to culture of *M. tuberculosis* from trunk washes (personal communication, Dr. Ray Waters, Orlando, Florida, May 2005). A gamma interferon assay is also under study.

Culture-positive elephants may have significantly lower values for A:G ratio, mean cell hemoglobin concentration, and glucose, and significantly higher values for platelets, band neutrophils, eosinophils, calcium, and bicarbonate.[109]

Tuberculosis is primarily a pulmonary disease; however, any organ system may become infected and extrapulmonary infection may occur alone or concurrently.[163] The lungs and thoracic lymph nodes are the main sites of pathology in elephants, with the appearance of lesions varying with the disease stage[267,229] (also Montali, unpublished observations, 1999). Firm, granulomatous, nodules, characteristically observed with less extensive disease progress to severe caseocalcareous and cavitating lesions in later stages. Pulmonary abscesses may form yielding opportunistic bacteria in addition to *M. tuberculosis*. Bronchial lymph nodes are typically enlarged. Pulmonary pathology may be extensive in advanced cases and mucopurulent bronchial plugs and mineralization may be seen. Epithelioid granulomas are observed histologically in the early stages and caseous, pyogranulomatous pneumonia is seen in the later stages. Acid-fast bacteria may be observed within caseated lesions in the lungs.[191]

**Differential diagnosis.** If clinical signs are present, dental disease, major organ dysfunction, and arthritis should be considered.

**Management.** Treatment regimens for elephant TB are based on protocols known to be effective in humans. In general, three antituberculosis drugs are administered for 2 months followed by two drugs administered for 10 months. First-line drugs include isoniazid (INH), pyrazinamide (PZA), rifampin (RIF), and ethambutol (ETH). Pharmacokinetic studies have been conducted in elephants, and data is available for INH,[57,164] EMB,[165] and PZA.[321] Further research is needed, however. Isoniazid and PZA may be given orally or rectally. Measurable blood levels for RIF have been achieved with oral administration only. Drug selection should be based on sensitivity results if possible. Suggested starting dosages are 5 mg/kg for INH (oral or rectal), 10 mg/kg for RIF (oral), 30 mg/kg for PZA (oral or rectal), and 30 mg/kg for ETH (oral). Ethambutol (bulk formulation) is rapidly expelled when administered rectally. Consult the guidelines for updated drug dosages and schedules. Blood levels of antituberculosis drugs should be measured to determine adequate absorption.[218,219] Some elephants have shown signs of toxicity (anorexia, lethargy, low-grade anemia, pica) at recommended drug dosages.

Elephants known to be shedding (or with a high degree of suspicion for the disease) should be isolated from other elephants, although the stress of social isolation must be given consideration because stress may exacerbate the disease.[24] After antituberculosis therapy is initiated, shedding generally ceases within a few weeks.

*M. tuberculosis* and *M. bovis* are very resistant to chemical disinfection. It is important to use agents that are specifically labeled "tuberculocidal" to clean elephant areas. Selected products are listed in Table 11.2; consult the Environmental Protection Agency (EPA) for a complete list of registered products. Products should be diluted and stored according to label specifications, and all product safety measures should be followed. The efficacy of a disinfectant is affected by numerous variables, including temperature, type of surface, product concentration, contact time, pathogen load, and the presence of organic material.

Tuberculosis organisms are sensitive to ultraviolet (UV) radiation, so maintaining infected elephants in open, sunny environments or under UV lights is advisable. If inside airflow can be controlled, it should be HEPA (High Efficiency Particle Arresting) filtered with >6 air exchanges/min.[163]

**Personnel safety.** Precautions should be taken to assure staff safety.[53] Exposure to infected elephants should be limited to essential personnel. Approved particulate (HEPA) filter masks that are rated to protect against TB and other protective clothing (gloves, coveralls, boots) should be worn. Hand washing is essential. The follow-

**Table 11.2.** Selected Tuberculocidal Disinfectants Available in the U.S.*

| Product | Contact Time (Minutes) | Active Ingredient | Manufacturer or Supplier |
|---|---|---|---|
| 1- Stroke Environ® | 10 | phenol | Steris Corporation www.steris.com |
| Clidox-S® | 5 | chlorine dioxide | Pharmacal Research Labs www.pharmacal.com |
| Ecotru® | 5 | parachlorometaxylenol | Envirosystems Inc. www.ecotru.com |
| Ultra Clorox Regular Bleach® | 5 | 6% NaOCL; mix 1 part bleach with 9 parts water | Grocery stores; www.clorox.com |
| Tek-Trol® | 10 | phenol | Valley Vet www.valleyvet.com and others |
| Virex TB® | 10 | ammonium chloride | Johnson Wax Products www.jwp.com |

*Follow label instructions on all products. For a complete list of EPA registered products, see www.epa.gov/oppad001/list_b_tuberculocide.pdf.

ing sources may be consulted for specific recommendations on protective equipment for humans:

1. OSHA TB Standards and Rules: www.osha.gov/SLTC/tuberculosis/standards.html 2.
2. Guidelines for Preventing the Transmission of *Mycobacterium tuberculosis* in Health-Care Settings, 2005 (CDC): www.cdc.gov/nchstp/tb/Federal_Register/New_Guidelines/TBICGuidelines.pdf.

## Anthrax

**Definition.** Anthrax (charbon, woolsorter's disease, milzbrand, splenic fever, malignant pustule, pustule maligna, black bane/bane) is a peracute or acute febrile infectious bacterial disease affecting most domestic mammals, many species of wild mammals and even birds (ostrich, gulls).[39] In elephants, the disease is seen primarily in free-ranging conditions, although there are reports of anthrax in zoo elephants in Bangladesh[206] and Britain.[263] Anthrax has worldwide distribution and was described in man over 3500 years ago.[89] It has been added to the list of emergency diseases in the United States, comparable to foreign animal diseases, because it is a zoonosis and has potential for use in bioterrorism.

**Etiology.** Anthrax is caused by an endospore-forming, aerobic, nonmotile, gram-positive rod *Bacillus anthracis*. The spores may remain in the soil for many decades. The pathogenesis of infection begins with ingestion of the vegetative form or spores. The organism enters the body through the lungs, intestines, or wounds on the skin. The organisms are engulfed by macrophages in which replication takes place until the macrophage bursts and allows spread of the infection throughout the body. More important, however, is that the vegetative cells begin production of a potent exotoxin that inhibits phagocytosis, increases capillary permeability, and interferes with the blood clotting cascade.[89]

**Epizootiology.** The epizootiology of elephant anthrax is basically the same as for other mammals. The vegetative form of *B.* anthracis is not highly competitive. If a carcass remains unopened, putrefactive organisms will soon destroy *B.* anthracis and spore formation will not occur. Although somewhat controversial, it is now generally believed that multiplication of the vegetative stage of *B. anthracis* occurs primarily within a living host.[89]

Elephants may contract anthrax via several pathways. Flies may carry the organisms on their feet and mouth parts from a carcass, contaminating any foliage they light upon (blow/bottle flies *Lucilia cuprina, Chrysomia marginalis, and C. albiceps*). Biting flies may inject the organisms (stable fly *Stomoxys calcitrans*, horsefly *Tabanus* spp.) or contaminate a wound.

When scavengers open up a carcass, organisms may be spread around the area.[155] When exposed to oxygen and desiccation, spore formation is rapid. Either the vegetative stage or spores may be moved on the feet and hair-coat of scavengers, or picked up by winds and aerosolized. Vultures often go to a waterhole following engorging on a carcass. Organisms may be on the vultures' feet or be excreted via the feces. Water runoff from rains or flooding may disseminate the spores far away to lowland areas causing a concentration of the spores when drying takes place. When elephants graze they often ingest the roots of the grass as well as the stems, which may enhance exposure to anthrax. See Figure 35.1 in the Africa section of Chapter 35.

Anthrax epizootics are seasonal and are usually associated with low-lying areas with high moisture and organic content, yielding an alkaline pH. An outbreak usually occurs during hot, dry weather following heavy rains or floods. In Namibia, anthrax occurs in November

in elephants, at the end of the dry season.[58,62,63] Animals congregate at water holes and overcrop the surrounding area, causing animals to eat closer to the soil.

Direct transmission from elephant to elephant is rare. However, in people, aerosol transmission from a person with fulminating pneumonia is a real possibility.[26] Anthrax was diagnosed in a person who was treating an infected tusk in an elephant with anthrax.[266]

**Clinical signs in elephants.** The clinical syndrome of anthrax in elephants has been described by many authors.[36,39,47,58,62,105,108,113,121,172,175,202,206,208,220,221,250,263,292,301,306] The severity of signs may be dependent on the degree of exposure and the source of the infection. Inhaled organisms produce pneumonia and dyspnea. Ingested organisms may cause colic and a severe hemorrhagic diarrhea. Edema and hemorrhage of the brain may cause ataxia, extreme listlessness, paralysis, and convulsions. Sudden death with no premonitory signs is characteristic of peracute anthrax. Early signs of acute anthrax (1–3 d course) include frequent urination, restlessness, and weakness of the hindquarters, with frequent falls.[210]

Following are the collective signs reported from an outbreak in a group of work elephants in Myanmar (Burma) during the mid-1800s: staring eyes, hemorrhages on the oral mucosa, bloody feces, shivering, trembling, leaning against trees, falling, inability to rise, ataxia, swollen trunk, failure to obey commands, swaying, convulsions, blindness, bloody discharges from body orifices, trunk paralysis, swelling between the front and hind legs and behind the ears, anorexia, dilated pupils, colic, and fever.

The exotoxin inhibits rigor mortis, but bloat and rapid putrefaction are characteristic following death, resulting in a "sawhorse" position of the carcass.[89]

**Diagnosis.** Clinical signs, rapid death, and condition of the carcass should alert the clinician to the possibility of anthrax. It is recommended that such carcasses not be opened; this prevents contamination of the surrounding soil with vegetative bacteria that may then produce endospores. There are no screening tests for anthrax.[15] The isolation of *Bacillus anthracis* is the primary definitive diagnosis. A PCR for *B. anthracis* is available also. This disease is a potent zoonosis, so caution should be used when conducting a necropsy or performing laboratory diagnosis. One must also be alert to the possible presence of nonpathogenic "boxcar"-shaped bacilli.

If a necropsy is conducted, there is blood exuding from body orifices, with marked subcutaneous edema, particularly around the upper neck and jaw. When tissue is incised, the blood is usually dark and thick and clots poorly. The primary lesions are the result of exotoxin damage to the reticuloendothelial system and vasculature. Widespread hemorrhages are seen on mucous and serous membranes of the heart and gastrointestinal system. Damage in subcutaneous areas causes extravasation of blood and fluids (swelling).

The spleen is usually enlarged, dark, and pulpy, which is caused by necrosis of the reticuloendothelial tissue. Either local or generalized edema may be seen in the muscle masses, subcutaneously, in the lungs, and even in the calvarium. Regional hemorrhagic lymphadenitis is seen particularly with inhalation anthrax. Ulcerative hemorrhagic enteritis may be seen with ingestion of the organisms.

**Differential diagnosis.** In warm climates, oleander *Nerium oleander* poisoning may produce peracute death, hemorrhagic diarrhea, and bloody fluid from the nares and mouth.[77] Peracute salmonellosis or other septicemic diseases should be considered, along with lightning strike, hyperthermia, and venomous snakebite.

**Management.** There is no treatment for anthrax in elephants. If anthrax is suspected, collect blood for culture, but leave the carcass unopened. The best management is to bury the carcass or burn it. Neither of these are easily accomplished in a field situation. Quaternary ammonium compounds are effective in destroying the vegetative form, but nothing short of incineration will kill the spores. If a carcass is handled in a diagnostic laboratory, insect control is crucial, and organs should be incinerated and the necropsy room thoroughly disinfected.

Mass vaccination was carried out in United States military troops during the wars in Kuwait, Iraq, and Afghanistan. The anthrax vaccine used (Anthrax Vaccine®) is a cell-free protein extract of cultures of *B. anthracis*; thus it is a killed-vaccine. A Sterne strain, nonencapsulated live spore vaccine (Anthrax Spore Vaccine,® Colorado Serum Co., Denver, Colorado) in a saponated diluent is used in animals.[297,295,296] The administration of this vaccine is not without risk in nonvalidated species. Three young llamas died following administration of an adult dose of the spore vaccine.[33] Oral vaccines have been used in elephants.[59,238]

## Salmonellosis

**Definition.** Salmonellosis (enteric epizootic typhoid, paratyphoid) is a bacterial disease presenting primarily as gastroenteritis, but occasionally the infection becomes systemic, producing a septicemia.[118,201]

**Etiology.** *Salmonella* spp. are small, straight, gram-negative, most motile, non–spore-forming rods. Genetically, all salmonella belong to the same species, but there are more than 2000 serovars/serotypes. Many serovars are named as if they were a separate species.[118] *Salmonella typhimurium* is one of the most widespread pathogens in the world.[201] Precise serovar typing with PCR technology was not generally available prior to 1995.

Serotyping is based on somatic antigens (O antigens), capsular lipopolysaccharide antigens, and flagel-

lar antigens (H-antigens), which help determine the virulence and invasiveness of a serotype. *Salmonella* spp. produce exotoxins and other substances that affect target cells in the intestine.

**Epizootiology.** The gastrointestinal tract of both warm and cold-blooded animals is the reservoir for salmonella organisms, which may be commensals and part of the normal flora of the gastrointestinal tract. The organisms are shed intermittently in the feces and may survive for months in soil, and will even multiply in water. Any contamination of feeds or water may allow ingestion of the organisms. Whether disease develops in the new host (elephant) depends on the infectious dose, resistance of the host to colonization within the gastrointestinal tract, and serovar of the salmonella.[118]

The pathogenesis of disease begins with destruction of epithelial cells in the terminal small intestine and proximal large intestine. This allows invasion of the organism into lymphoid cells in the submucosal area. If the host's resistance mechanism contains the infection to this location the result is diarrhea. If the salmonella serovar has the necessary virulence factors, the organism may proliferate in the macrophages of the spleen and liver and disseminate to produce systemic infection and septicemia.

**Signs in elephants.** Clinical outbreaks of salmonellosis usually follow immune depression from stress (overcrowding, transporting, parturition), failure of passive transfer, postsurgical cases or viral diseases.[32,56,88,131,150,167,171,176,179,194,214,237,264,307,315,316,322] Anorexia, lethargy, diarrhea, and colic are the initial signs. With systemic invasion, septicemia and toxigenic shock occur.[38,102] The collective signs observed in four African elephants included anorexia, lethargy, severe diarrhea, colic, abortion, and vaginal discharge. The course of the disease lasted only 3 days in three cases but many months in the other.[65]

Atypical salmonellosis appeared in a 6-year-old African elephant. Initially anemia was the primary concern with PCV levels of 7–17 %, coupled with a neutrophilia and a regenerative left shift. Other signs included ventral edema, weakness, reluctance to lie down, intermittent anorexia, and minimal diarrhea.[236] Medications failed to aid the elephant, and on day 17 following recognition of disease the elephant was euthanized. *Salmonella typhimurium* was cultured from the liver and spleen, which were grossly and histologically necrotic. This elephant never exhibited icterus. No source of the infection was determined.[236,99]

*Salmonella typhimurium* was isolated from feces of an 18-year-old female elephant at a midwestern zoo. Signs included colicy signs, formed fecal balls coated with mucus, inappetence and lethargy.[279] She was treated with trimethoprim sulfa intravenously and recovered.

Four of six young (2–4-year-olds) Asian elephants transported from India to a traveling circus in Germany died within 3 weeks of arrival of acute gastroenteritis. *Salmonella dublin* was cultured from internal organs at necropsy. The stress of travel certainly had a bearing on these cases.[88]

The Basel Zoo experienced two outbreaks of salmonellosis in African elephants. In 1988 a cow elephant became extra nervous and ate little. She became apathetic and was immobilized to examine the teeth. Retained segments of a front cheek tooth had caused gingivitis. After the immobilization, she developed diarrhea and *salmonella enteriditis* was cultured from the feces. Treatment was of no avail and she died on day 8. Three other elephants in the zoo had positive cultures for *Salmonella enteriditis*. These were treated prophylactically with neomycin sulfate and fecal cultures became negative. In 1996 an elephant was immobilized to treat a tusk pulpitis. One month later she became apathetic and had a poor appetite. Salmonellosis was suspected and *Salmonella typhimurium* was cultured from the feces. Cultures were obtained from seven other elephants in the elephant house. All were positive for *Salmonella typhimurium.* All were treated with antibiotics and none of these elephants died. No source for the infection was determined, but rodents were suspected as the reservoir.[214]

**Diagnosis.** In the live animal with gastroenteritis, the feces is cultured. Keep in mind that shedding may be intermittent, so a single fecal culture does not rule out infection. If septicemia is evident, blood should be cultured. At necropsy the spleen, liver, and bone marrow are good tissues from which to obtain cultures. It is necessary to use selective media to culture salmonella from feces. Each laboratory has its own favorite media for this task, but MacConkey agar, brilliant green agar, or selenite F broth are used. Samples from tissue at necropsy may be plated directly onto blood agar. Serotyping is done using DNA probes and primers for PCR technology.[118,194]

**Management.** This is essentially the same as for many enteric infections. Rapid correction of electrolyte imbalance and control of diarrhea are essential. Sodium bicarbonate is administered to correct the metabolic acidosis. Administration of potassium chloride and other electrolytes is crucial. The infection must be brought under control quickly with antimicrobial agents determined by culture and sensitivity testing. Antibiotics will not sterilize the intestine of salmonella organisms, but may give sufficient control to allow the animal's immune system time to respond and control the disease.

It may be desirable to supplement or replace the gastric flora with a product such as Lactobio-s® (contains *Lactobacillus acidophilus* and *Streptococcus faecium*; RX Veterinary Products, Kansas City, Missouri, USA).

No vaccines are available.

## Colibacillosis

**Definition.** Colibacillosis is a group of diseases caused by pathogenic strains of *Escherichia coli*. It may be an invasive bacterial infection or an enterotoxemia. Nonpathogenic strains are often found as normal flora in the large intestine of animals.[116]

**Etiology.** *Escherichia coli* is a ubiquitous, gram-negative, motile, non–spore-forming enteric bacterium. There is only one species, *E. coli*, but there are hundreds of strains. Because it is a normal inhabitant of the large intestine, it may be difficult to diagnose colibacillosis without conducting laboratory diagnostic tests to identify the strain isolated.[34]

This author is not qualified to discuss the details of *E. coli* strains, but a basic knowledge is necessary to understand the clinical disease and how it is manifested in different species. The reader is referred to the references for more details. Strains are classified based on capsular lipopolysaccharides and flagellar antigens, which modify virulence and potential invasiveness.

Pathogenic strains of *E. coli* excrete at least five products of medical importance; enterotoxins, siderophores, shigalike toxins, cytotoxic/necrotizing factors and hemolysins.[116] The enterotoxins interfere with water and electrolyte balance so that sodium, chloride, and bicarbonate ions flow into the intestinal lumen. This leads to diarrhea, hypovolemia, and metabolic acidosis. If acidosis becomes severe, hyperkalemia develops. Siderophores aid *E. coli* to remove protein-bound iron from the host and incorporate the iron as a necessary ion for multiplication. Shigalike toxins inhibit protein synthesis. Hemolysins damage cell membranes and aid *E. coli* to become more virulent and invasive.

**Epizootiology.** *E. coli* has a worldwide distribution as part of the normal flora of many species of animals. The transmission of the organism from one animal to another is via the fecal-oral route.

**Clinical signs in elephants.** There are multiple forms of colibacillosis.[166,298] One causes an enterotoxic diarrhea. Another produces an invasive disease, which results in generalized infection and septicemia. A third form is a non-enterotoxigenic diarrhea. Various forms of colibacillosis are caused by different strains. Some may be pathogenic only for certain species, such as the one causing edema disease of swine. A human pathogenic strain (O157:H7) is of great public health concern, particularly when found in meat that has not been adequately cooked.[116]

*E. coli* has been isolated sporadically from uterine infections, foot abscesses, and septicemias. In almost all of the reports of *E. coli* from elephants, it appears that the organism was a secondary invader, a contaminant or a coinfectious agent with other enteric bacteria, or beta hemolytic streptococci.

**Diagnosis.** Clinical signs and lesions are not diagnostic. Isolation and identification of the organism and strain is necessary for a confirmed diagnosis.[34] Colibacillosis may also be seen as a secondary infection accompanying such diseases as enterotoxemia caused by *Clostridium perfringens*.

The techniques employed to identify pathogenic strains of *E. coli* vary with the type of disease exhibited. If enterotoxigenic strains are suspected, it is necessary to identify certain fimbria (K88 or K99) antigens, which is done using ELISA technology, or at necropsy, quantitation of *E. coli* in the small intestine (not a normal site for *E. coli* habitation as part of the normal flora). IFA may be used to demonstrate the toxin. DNA probes and PCR primers are used to identify specific toxins.

For invasive strains, culture of the organism from what should be sterile sites such as joints, bone marrow, spleen, or blood is definitive.

**Management.** Rapid correction of electrolyte imbalance and control of diarrhea are essential to saving the life of an infected animal. Sodium bicarbonate is administered to correct the metabolic acidosis. Administration of potassium chloride and other electrolytes is crucial, but these should be monitored so that proper amounts may be calculated and administered in the fluid. The infection must be brought under control quickly. Antimicrobial agents such as gentamicin, amikacin, trimethoprimsulfa, or ceftiofur may be initiated pending antimicrobial sensitivity results. The administration of neomycin orally to combat infection in the small intestine (neomycin is not absorbed from the intestine but will act locally) will help correct the electrolyte flow into the lumen.

Vaccines are available for protection of specific serotypes in cattle, horses, dogs, and cats. There are no reports of vaccine usage in elephants.

## Mycoplasmosis

**Definition.** Mycoplasmosis is an infection of mucous or serosal membranes which may cause an autoimmune reaction in joints.

**Etiology.** *Mycoplasma* spp. are small bacteria that lack a cell wall. Many are host specific. Another closely related organism is *Ureaplasma* spp., which has been isolated from elephants.

**Epizootiology.** *Mycoplasma* spp. are found worldwide and may be restricted in their distribution according to the distribution of their primary host(s). They may be part of the normal flora of mucous membranes. Transmission is by direct contact or ingestion of feed and water recently contaminated by an infected animal.

**Clinical signs in elephants.** Mycoplasmosis is usually a genital/urinary tract infection with minimal signs evi-

dent. The arthritic response was presumed to be a mycoplasmal hypersensitivity mechanism of the delayed type.[136] There was a good deal of investigation carried out over a 10-year period with 120 elephants, indicating that mycoplasmas were associated with a rheumatoid type of arthritis characterized by shifting leg lameness, which was exacerbated by prolonged rest or cold weather.[40,43,44,45,42,284] Other signs included swollen and warm joints, general weakness, lethargy, behavioral changes, and weight loss, in spite of a good appetite. There was an elevated immunoglobulin level in the blood with a rise in rheumatoid factor and other immune antibodies. Elephants had a rise in CF antibody titer, particularly at the time of a flare-up.[44,41] Many of the elephants studied responded to antimycoplasma antibiotic therapy.

Conclusive evidence as to the cause of rheumatoid arthritis in elephants has not been reported in the literature.

**Diagnosis.** Showing that *Mycoplasma* spp. or *Ureaplasma* spp. are the cause of a specific disease may be difficult because this organism may be a normal inhabitant of mucous membranes. In elephants, *Mycoplasma genitalium* has been isolated from the genital mucous membranes. *Mycoplasma* spp. are fastidious, and specimens should be transferred quickly to a suitable transport medium or onto appropriate media for isolation and identification of mycoplasmas. Not all laboratories are equipped for mycoplasma identification.

Immunofluorescent antibody testing may be used to identify mycoplasmas in frozen or fixed tissues. DNA probes and PCR technology are now adapted for identification of species or strains. Serologic tests include CF and indirect hemagglutination.

**Management.** Mycoplasmas are generally sensitive to tetracycline antibiotics. Elephants have been treated effectively with long-term, but intermittent, doxycycline therapy. No vaccines are available.

## Minor Infectious Diseases

These are diseases that rarely produce clinical disease in elephants or produce only an antibody response; they are tabulated in Tables 11.3 and 11.4. A few diseases may be discussed because of their importance in differential diagnosis with more important diseases.

### Rinderpest.

Definition. Rinderpest is a highly contagious disease of artiodactylids characterized by fever, lymphocytopenia, erosive stomatitis, gastroenteritis, and diarrhea.[16,48,248,262]

Etiology. Rinderpest is caused by a virus in the family Paramyxoviridae, genus *Morbillivirus*, closely related to the viruses that cause canine distemper and measles. Several different strains vary in virulence, but all are immunologically indistinct.[181,262]

Epizootiology. Elephants may come into contact with rinderpest in ruminants, but since this is not a clinical disease in elephants, nothing is known about the transmission of the organism to elephants causing an antibody response.

In ruminants, transmission of rinderpest is by direct contact. All animal discharges contain the virus, and the infection route is either via the respiratory tract or by ingestion. No carrier state of rinderpest has been established in cattle. Clinically affected animals shed the virus for 2–3 weeks if they survive.

Clinical signs in elephants. This is not a clinical disease in elephants.

Diagnosis. A number of serologic tests have been used to detect antibodies in ruminants and elephants, including ELISA and virus neutralization. Isolation of the virus in tissue culture, PCR, AGID, IFA, IHC, or animal inoculation is required for definitive diagnosis in artiodactylids.[248] No virus has been isolated from elephants.

### West Nile viral encephalitis.

Definition. West Nile viral encephalitis is a mosquito-borne viral meningomyeloencephalitis affecting a broad host range, including humans, horses, camelids, and a large number of bird species. The entire host range is unknown, but elephants have sero-converted, so WNV should be considered a potential emerging disease in elephants.

Etiology. The etiology is an arbovirus (WNV) that is closely related to the viruses that cause St. Louis encephalitis, Japanese encephalitis, and Murray Valley encephalitis.

Epizootiology. Although the disease was first identified in crows in the United States, many other species of birds may serve as natural or amplifying hosts. Transmission is via mosquito bites (*Culex, Aedes, Anopheles* spp.). The disease has been recognized in Africa and the Middle East for many years and was first diagnosed in the United States in 1999. The disease has become endemic and is spreading throughout the United States.

Signs in horses and humans. To date, clinical disease has not been definitively documented in elephants, so the syndrome in other species is described. Two suspected cases of WNV are discussed in Chapter 24.

The incubation period is 6–10 days. Most infections are subclinical in horses and humans, but 10% of affected individuals develop a fever and the encephalitic signs of depression, ataxia and paresis, particularly of the hindlimbs. More advanced signs include head shak-

**Table 11.3.** Microorganisms Causing Antibody Response but No Clinical Disease in Elephants

| Disease | Etiology | Epizootiology | Diagnostic Procedures | Comments | References |
|---|---|---|---|---|---|
| Bluetongue | *Oribivirus* sp. Family Reoviridae | Worldwide distribution, transmission by *Culicoides* spp. midges. | Inoculation of ECE and TC<br>Serology: AGID, CF, ELISA, IFA, VN, HI | Antibody response but no clinical disease. | Formentry 1994, MacLachlan 2004, Mehrotra 1990, Stott 2001 |
| African horse sickness | *Oribivirus* sp. Family Reoviridae | Transmission by *Culicoides* midges. Endemic areas in Africa, Middle East, & parts of Asia. | VI. Serology: CF, AGID, IFA, VN, HI | Numerous serologic responses in elephants, but no clinical disease. | Barnard 1995, Binepal 1992, Davies 1997, Meiswinkel 1994 |
| Rinderpest | *Morbillivirus* sp. Family Paramyxoviridae | Transmission via contact with animal discharges by inhalation or ingestion. | TC, PCR and animal inoculation. Serology: AGID, IFA, ELISA, VN | Once the scourge of Africa; now close to extinction because of vaccination. Serologic reports in elephants, but no disease. | Bhat 1997a, Mehrotra 1990 |
| Japanese encephalitis | *Flavivirus* sp. Family Flaviviridae | Transmitted by mosquitos, *Culex* sp. Found in temperate and tropical Asia. | VI from CSF, TC. Serology: HI, CF, RIA, VN, ELISA | A zoonosis; only one report of a serologic titer. | |
| Influenza, type A | *Orthomyxovirus*, type A Family Orthomyxoviridae | May be transmitted by ingestion or inhalation. Waterfowl may be a reservoir. | VI on TC and ECE. Serology: HI, NI | Titer in an elephant reported only once.<br>Influenza type A is important in chickens, swine, horses, and marine mammals.<br>Potential serious zoonosis. | Schroder 1992 |
| Plague, Black Death | *Yersinia pestis* | Infection-tolerant rodents are the reservoir in endemic areas.<br>Transmitted by fleas.<br>A significant zoonosis, which may spread without fleas, when in people. | BC, animal inoculation Serology: HA, HI, ELISA | Only one serologic response reported in elephants. | Gordon 1979 |

ing, incessant chewing, paralysis of the lower lip or tongue, severe ataxia, ascending paralysis, and terminal recumbency.

In humans, the virus can cause fever and severe headaches and may cause serious illness in people over 50 years of age, including swelling of the brain and spinal cord. Between 1999 and 2001 there were 149 cases in the United States, with 18 deaths. During 2002 alone there were 4,156 cases with 284 deaths—so the problem is growing. West Nile virus disease is now in every state in the contiguous United States.

Signs in elephants. Signs are presumed similar to those seen in the horse.

Diagnosis. Several serologic assays are used to diagnose WNV infection, including serum neutralization tests and IgM-ELISA. Perivascular cuffing with lymphocytes and histiocytes is observed on brain tissue histologically. Immunohistochemistry may be used to confirm the presence of WNV antigens in tissues.

Differential diagnosis. Many diseases have a neurologic component that could pose a problem in differential diagnosis, including rabies, head trauma, lead poisoning, brain abscess, plant poisoning, brain tumor, toxicosis, hydrocephalus, and insecticide poisoning. WNV occurs seasonally associated with the mosquito season.

Management. Mosquito control is critical. Two vaccines are licensed for use in horses. It has been used in a variety of zoo mammals with serologic titers recorded that are protective in the horse. General nursing care should be provided.

**Table 11.4.** Minor Infectious Diseases of Elephants

| Disease | Etiology | Signs | Diagnosis | References |
|---|---|---|---|---|
| Necrobacillosis (necrotic laryngitis, calf diphtheria) | *Fusobacterium necrophorum, Haemophilus somnus* | Laryngeal ulcers, diphtheritic membrane on larynx and trachea, dyspnea, coughing, salivation, fever, malodorous breath, septicemia. | Signs, culture of the organisms. | Sailer 1951 |
| Cowdriosis (heartwater) | *Cowdria ruminantium* | No signs noted in elephants. | Organisms seen in brain of an elephant that died of another disease. | Okewole 1993 |
| Staphylococcosis (botryomycosis) | *Staphylococcus aureus* *Staphylococcus* spp. | Abscesses, localized suppurative inflammation. Exudate creamy with no odor. | Culture of gram-positive cocci from inflamed tissue or abscess. Cocci in clusters. | Sleeman 2003, Mehrotra 1999 |
| Streptococcosis | *Streptococcus zooepidemicus, Streptococcus* spp. | Infection may be secondary to other infections of many organ systems. | Culture of gram-positive cocci from inflamed tissue. | Verge 1934b |
| Pseudomoniasis | *Pseudomonas aeruginosa, Pseudomonas* spp. | Often a secondary infection or a mixed infection in the foot or other abscesses. | Culture of the organism from infected tissue. | Sutopa 1998 |
| Pasteurellosis (hemorrhagic septicemia) | *Pasteurella multocida, Pasteurella* spp. *Mannheimia haemolyticum* | Dyspnea, coughing, weakness. | Culture of the organism from the nares or lung tissue. May be a commensal. | Singh 2002, Thiruthalinathan 1995, Wickremasuriya 1982, De Alwis 1965, Evans 1906 |
| Mycoplasmosis | *Mycoplasma* spp. *Eureaplasma* spp. | Shifting leg lameness, swollen, warm joints, general weakness, reluctance to move. A type of rheumatoid arthritis? | A rise in CF antibody titer during a flare-up. Response to therapy. Organism is fragile, cultures quickly. | Kirchhoff 1996, Clark 1976, 1979, 1980, 1981 |
| Papillomatosis (warts) | Papillomavirus | Some reports of papillomas proved to be nodules caused by herpesvirus. Other reports indicate isolation of papillomavirus. | Virus isolation. Differentiate from herpesvirus. Not fatal. | Sironi 1990, Sundberg 1981, 1997, 2001, Von Hegel 1989 |
| Corynebacteriosis (abscesses) | *Arcanobacterium* (*Actinomyces*) *pyogenes*); formerly Corynebacterium | Abscesses and pyogenic inflammation of organs or tissues throughout the body. No odor to exudate unless it is a mixed infection. | Culture of small, gram-positive rods. It is a facultative anaerobe. | |

**Clostridial diseases.** Clostridial diseases are ubiquitous, but they occur only sporadically in elephants (see Table 11.5). *Clostridium* spp. are gram-positive, rod-shaped, anaerobic bacilli. All form spores that may persist in the soil for months or years. Some of these organisms may be found in the normal flora of the digestive tract and become pathogenic only if accessible tissue is damaged as a result of deep penetrating trauma to the muscle bundles or a compromised gastrointestinal mucosa.

Clostridial organisms produce potent exotoxins that are primarily responsible for the disease these agents cause. The toxins are metabolites that are produced as the organism grows in the host tissue, except for botulinum toxin, which may be ingested preformed. A given organism may produce single or multiple toxins, each with a different effect on the host. Botulinum and tetanus toxins are neurotoxic only. Most other clostridial organisms produce toxins with both local and systemic effects, including hemolysis and local tissue necrosis.[4]

Botulism. *C. botulinum* is distributed worldwide. One or more of the types (A–G) is probably toxic to all vertebrates. Only two cases have been reported in elephants, but clinical diagnosis is difficult and may have been simply overlooked.[64,83] There is good reason to believe that elephants may be susceptible.

The syndrome is believed to be similar to that seen in most other mammals, consisting of a progressive paralysis of all skeletal muscles. Initially, incoordination,

**Table 11.5.** Clostridial Diseases of Potential Concern for Elephants

| Disease | Etiology | Signs | Differential Epizootiology | Pathology | Management | References |
|---|---|---|---|---|---|---|
| Botulism | *Clostridium botulinum* | Flaccid muscle paralysis | Ingestion of preformed toxin. | None. | Specific antitoxin, support of respiration. | Elze 1962, Garlt 1977 |
| Tetanus | *Clostridium tetani* | Muscle spasms, tetany | Infected teeth, anaerobic wound contaminated with spores. | None. | Antitoxin, muscle relaxants. Prevent with toxoid vaccination. | Burke 1975a, b; Goss 1942 |
| Malignant edema | *Clostridium septicum* | Edematous swelling around a wound. No gas, sudden death. | Contamination of a wound with spores of the organism. | Edematous subcutaneous cellulitis. | Broad-spectrum antibiotics. Prevent with vaccination. | |
| Enterotoxemia, type C | *Clostridium perfringens,* type C | Sudden death, severe diarrhea, gas colic, prostration | Unknown in elephants. | Hemorrhages of the mucosa of intestine, which is distended with gas and fluids. | Prevent with vaccination. | Bacciarini 2001 |
| Enterotoxemia, type D | *Clostridium perfringens,* type D | Sudden death, convulsions, circling posterior paralysis, minimal diarrhea | Unknown in elephants. | Hemorrhages on serosa of intestine, epicardium, and endocardium. No gas. | Prevent with vaccination. | |
| Blackleg | *Clostricium chauvei* | Fever, hemorrhagic swelling and gas formation in muscles | Unknown, only one case reported in elephants. | Hemorrhage and gas in muscles. | Prevent with vaccination. | Prescott 1971 |

muscle weakness, and recumbency are seen, leading finally to flaccid paralysis of all muscles, including respiratory muscles. Body temperature is not elevated. The pupils of the eyes become dilated. Salivation is decreased, and mucous membranes become cyanotic.[64,83]

Because *C. botulinum* may be cultured from a normal digestive tract, isolation of the organism is not diagnostic. A definitive diagnosis can be made only by the injection of filtrates of suspected feed materials or gut contents into mice or guinea pigs. Control animals are given simultaneous injections of protective doses of specific antitoxin.

After signs have developed, little can be done other than to support respiration. Antitoxins (toxin-type specific) used to treat human cases are not available for animals.

No toxoid bacterins against botulism are available for protection of animals or humans.

Tetanus. *Clostridium tetani* occurs worldwide as a soil saprophyte, but it can also be found in the feces of horses, humans, and cattle. Tetanus is more common in tropical regions than in cold climates. There is wide variation in the susceptibility of animal species to tetanus toxin. Horses, nonhuman primates, and swine are highly susceptible, with cattle, sheep, goats, and humans less so and dogs and cats quite resistant. It is not known about resistance or susceptibility in elephants.

Tetanus develops when wounds have been contaminated with soil or feces containing *C. tetani* spores. Contaminated deep wounds with devitalized tissue are most at risk of harboring the organism.[69,120,308] Such wounds are poorly aerated, providing optimum conditions for growth of the anaerobic organism.

Clinical signs seen in elephants are reported by Burke[27,28] and Goss.[98] The latter author's report follows. An 8-year-old female was unable to open her mouth. She was hypersensitive to noise and touch, becoming tense and raising her tail. Her body temperature was 98.6°F (keep in mind that if tetanic spasms are present the body temperature may be elevated).

Although there were several cracks around the toenails, none of the wounds were thought to be anaerobic. 100,000 units of tetanus antitoxin (TAT) were administered and the next day she seemed somewhat relaxed. However, on the third day she was found in lateral recumbency and in tetanic spasms. A sedative (112 grams of chloral hydrate per rectum) was administered. Periodic sedation was necessary to keep her relaxed. She was raised to her feet with a sling and left in the sling

overnight. On the fifth day she was unable to stand without the sling. Over the previous 5 days 360,000 units of TAT were administered subcutaneously. The elephant was kept in a sling and force-fed a slurry of bran mash through a stomach tube for 29 days, at which time she began to masticate and swallow feed.

Debride and cleanse any visible wound. Tetanus antitoxin should be administered at a dose of 225 units/kg body weight, half intravenously, the other half intramuscularly. Anaphylactic shock is a hazard of this therapy because tetanus antitoxin is a horse serum product. Be prepared to administer epinephrine. Broad-spectrum antibiotics should be administered to kill organisms that may not be reached with wound cleansing. The elephant should be placed in a nonstimulating environment and tranquilized as appropriate.

Supportive care is crucial to success. Be prepared to sling the elephant. Water may be administered by rectal lavage. For food, the author uses a slurry of quick-cooking rolled oats. The quantity of the breakfast cereal selected is put into boiling hot water, allowed to cool, and then diluted to a consistency that may be pumped through a stomach pump.

Tetanus toxoid vaccines are readily available. See Chapter 7 for vaccination recommendations.

Malignant edema. Malignant edema (gas phlegmon, gas edema, bradsot, braxy) occurs worldwide in a broad host range, including domestic livestock, horses, humans, elephants, dogs, and cats. *C. septicum* produces a toxin that causes severe edema. It is basically a soil organism, but it has been found in both spore and vegetative form in the intestines of healthy animals.

The organism invades tissue through a necrotic, deep wound that provides anaerobic conditions. Oral wounds and bruises are common entrance sites. The organism may also gain access to body tissue from disruption of the stomach epithelium. As the organism grows, toxins are produced and clinical signs appear within 1 to 3 days. Two types of syndromes develop. One is the typical wound infection and edema. The other is an acute systemic disease similar to braxy of sheep. With wound infections, clinical signs include a rapidly spreading, edematous swelling in the subcutaneous tissue surrounding the wound. Little, if any, gas forms in this disease, in contrast with blackleg. Other signs include fever, rapid pulse, anorexia, depression, and weakness. C. septicum affects animals of all ages. Death may occur 12 days after signs develop.

Other clostridial diseases. Other clostridial diseases reported include blackleg *Clostridium chauvei*[231] and enterotoxemia *Clostridium perfringens*.[4,92]

## Leptospirosis

Leptospirosis is a common disease in many species of domestic animals caused by one or more serovars of *Leptospira* sp. Prior to a well-documented case of leptospirosis in a 22-year-old female Asian zoo elephant (personal communication, Dr. Rita McManamon, Atlanta, Georgia, June 15, 2005) it was presumed that elephants could develop a positive titer to one or more serovars of leptospira, but clinical disease did not occur.[18,151]

Clinical signs in the case mentioned appeared first as a chronic weight loss (from 3334 kg [7350 lb] to 2903 kg [6400 lb]) over a 4-month period. Anorexia was profound. Leptospirosis was included in the differential diagnosis when the elephant developed uveitis and hypopyon. Titers for multiple serovars of leptospira reached 1:12,800.

The liver was the organ system infected. Icterus was marked. The sclera and hypopyon were both bright yellow. Total bilirubin reached 9.4 mg/dl, and liver enzymes were elevated. Ventral edema became pronounced, accompanied by ulcerating lesions of the vulva and various areas of the skin. The tip of the tail necrosed from vasculitis.

Blood urea nitrogen and creatinine levels remained normal throughout the course of the disease, indicating that the urinary tract was not involved.

Diagnosis was based on elevated titers for leptospira serovars plus hypopyon and uveitis. The organism was not isolated nor could antigens be detected by PCR evaluation. Two other elephants cohabitating with the ill elephant developed low titers (1:200–400) for *Leptospira icterohemorrhagica,* but they did not develop clinical disease. The zoo elephants were maintained on a standard protocol for monitoring for *Mycobacterium tuberculosis*. No positive cultures have been detected at this zoo.

Early treatment consisted of tetracycline. When leptospirosis was considered, a newer form of tetracycline (doxycycline) was begun and, later, enrofloxacin was added to the therapy. Appetite improved and weight is being regained. The ulcerative dermatitis responded to the antibiotic therapy and topical medication.

Another case was diagnosed in a 6-year-old Asian elephant in India (personal communication, Dr. Arun Zachariah, Kerala, India, July 6, 2005). This young elephant died of nephritis and adrenocortical infection.

## Pasteurellosis

Pasteurellosis (hemorrhagic septicemia) refers to a variety of localized and/or systemic diseases, frequently involving the respiratory system. The organisms have worldwide distribution and may be found as part of the normal flora of many animals.[21]

The two most common organisms involved in animal infections are *Pasteurella multocida* and *Mannheimia (Pasteurella) haemolytica*, which are small, pleomorphic, gram-negative rods or coccobacilli. They are nonmotile, facultative anaerobic and may exhibit bipolar staining with Giemsa or Wright's stain.[195]

Transmission is via aerosol spread or by ingestion of

contaminated feed and water. Insect and tick transmission may occur, and the organism may enter through contaminated wounds. These are generally thought to be opportunistic pathogens, and some predisposing factor such as trauma, stress, or intercurrent disease contributes to the development of clinical disease. Young animals are frequently victims.

Pasteurellosis has been reported in elephants, but only sporadically.[55,68,253,271,287] *Pasteurella* spp. has been isolated from abscesses and foot infections.[80] Pneumonic forms have also been reported.

Diagnosis is by isolation and identification of *Pasteurella* spp. or *Mannheimia haemolyticum* combined with a disease presence. Because pasteurellosis may be secondary to other disorders, consider that another agent may be a more important factor (Miller 2001b). Serotyping, genomic fingerprinting, and PCR-based assays may be used to characterize isolates when investigating an epizootic.[195]

The form of disease caused by pasteurellosis varies, so many other diseases must be included in a differential diagnosis, including trauma, foreign-body reactions, staphylococcosis, salmonellosis, and pneumonia caused by various agents.

Sanitation and minimizing stress are necessary to prevent pasteurellosis. Antimicrobial therapy based on sensitivity testing may be necessary to combat systemic infection. Vaccines are used in some species, including wild animals, but no such use has been reported in elephants.

**Staphylococcosis.** Staphylococcal infections are no different in the elephant than in other domestic and wild animals. *Staphylococcus aureus* is one of the more common organisms isolated from abscesses. *S. aureus* is a large, gram-positive coccoid organism that is frequently found in clusters (like grapes) on direct examination of the exudate. They are opportunistic pathogens that require some damage to the skin or mucous membrane to initiate infection (insect bites, wounds). The organisms are found worldwide.

Abscesses of various sizes are seen in elephants. The exudate is usually whitish and creamy with no odor. A localized granulomatous lesion caused by staphylococcal infection is called botryomycosis (originally thought to be a fungal infection). An elephant developed botryomycosis of the mammary gland, which ultimately had to be amputated.[275] Septicemia has been reported.[183] Secondary infection with staphylococcal organisms may be seen accompanying other diseases.

The thickness of the skin makes palpation difficult for abscesses in elephants. Aspiration of exudate with a 16 gauge needle makes diagnosis possible before rupture through the skin. Culture of the organism from abscesses or other suppurative lesions is definitive.

Differential diagnosis includes abscesses caused by other organisms, tuberculosis, seroma, and hematomas.

External abscesses may be brought to maturity with hot packs or counterirritant agents and then surgically lanced to provide ventral drainage. Daily irrigation of the abscess with disinfectants such as povidone iodine solution hastens healing by keeping the tract open. Antimicrobial therapy should be based on sensitivity tests.

**Abscesses and foot infections.** Abscesses, wounds, and foot infections are covered elsewhere in the book. However, the microorganisms isolated from these lesions are listed in Chapter 20, Table 20.4. Note that elephants form granulation tissue quickly, which produces a large amount of a thick, cream-colored, odorless exudate. The granulation tissue and exudate may occlude the tract to an abscess or foot infection and prevent proper drainage.[80,135] Regional venous profusion with antibiotics has been reported for osteitis.[277]

**Fungal infections.** We know little about the normal fungal flora of the mucosa of the gastroenteric system, respiratory system, oral cavity, nasal cavity (trunk), urogenital system, conjunctival sacs, external ear canal, and/or even the skin surfaces. A study reported two fungal species being isolated from the elephant colon *Piromyces mae* and *Piromyces dubonica*. These are probably commensals.[153]

A number of fungal microorganisms have been isolated from the skin,[147,203] corneal ulcerations (*Aspergillus* sp. *Cladosporium c*urvularia),[137] external ear canal,[147] or other tissues of elephants. These are not a major cause of disease in elephants. None of the common dermatophytes of domestic livestock and pets (*Trichophyton* spp., *Microsporum* spp.) are reported in elephants. Three dermatophytes have been isolated from hyperkeratotic, crusty skin lesions of elephants (*Trichothecium* sp., *Scopulariopsis* sp., and *Aspergillus* sp.). Lesions caused by T*richosporiella* sp. have been treated and cured.[126] One yeast infection is reported.[104]

A number of fungi produce mycotoxins that may have a profound effect on animals consuming forage contaminated by the fungus. No mycotoxicoses have been reported in elephants, but one should avoid feeding moldy feeds to elephants in captivity. Two fungi that should be of concern include *Pithomyces chartarum,* which produces sporidesmin, the cause of facial eczema in sheep in New Zealand. The fungus grows on rye grasses (*Lolium* spp.). The other is *Aspergillus flavus*, commonly growing on corn, small grains, and peanuts and producing aflatoxins.

## BIOTERRORISM

Bioterrorism is currently uppermost in many people's minds. Veterinary clinicians should have a clear picture of the disease agents that may be used, because they are most likely to be the first to observe evidence that the agents are in the animal population.

The Centers for Disease Control and Prevention (CDC) in Atlanta, Georgia, has been designated as the lead agency in the United States in attempting to prevent devastating loss to human beings and animals.[54] Six disease agents have been classified in Category A as the following: "having the greatest potential for inflicting large numbers of human casualties, can be manufactured and disseminated on a large scale, require significant efforts in public health preparedness and are most familiar to the public."[54] Diseases in category A include anthrax *Bacillus anthracis*, botulism *Clostridium botulinum*, plague *Yersinia pestis*, smallpox *Variola major*, tularemia *Francisella tularensis,* and hemorrhagic fever (Ebola virus). Five of those diseases pose a risk to domestic animals. Two of the diseases have been diagnosed in elephants.

Category B diseases pose less risk, but nonetheless must be considered. These include brucellosis *Brucella* spp., glanders *Burkholderia mallei*, melioidosis *Burkholderia pseudomallei*, psittacosis *Chlamydophila psittaci*, endemic typhus fever *Coxiella burnetii*, Q fever *Rickettsia* prowazelii, viral encephalidites (Eastern, Western, Venezuelan), toxins (icin *Ricinus communis*, staphylococcal enterotoxin B), salmonellosis *Salmonella* spp., colibacillosis *Escherichia coli* (Strain O157:H7), and cryptosporidiosis *Cryptosporidium* spp.

Category C diseases are emerging diseases such as Nipah virus (pigs and humans), Hantavirus (rodents and humans), and West Nile fever (West Nile virus, birds, mammals, elephants, mosquitos and humans).

This is not meant to be a scare tactic. It is simply a reality in the world we live in during the 21st century. Clinicians must remain vigilant and be prepared to report any suspicious clinical signs or diseases quickly. Additional information on bioterrorism and fact sheets and Powerpoint lectures on 116 reportable and zoonotic diseases are available at www.cfsph.iastate.edu.

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# 12 Parasitology

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## INTRODUCTION

This chapter is not meant to be a definitive discussion of all the ramifications of the parasites of elephants; however, sufficient information will be discussed to allow general identification, describe the life cycle (if known), make a diagnosis of parasitism, and aid in the management of parasitism in elephants. The taxonomic classification of parasites is in constant flux; therefore, a recent reliable publication should be selected as a source for taxonomic information. In this book, the classifications of Levine[62] for protozoa, Yamaguti and Bowman,[13,117] and Van de Westhuysen[110] and Lapage[61] for helminths, and Zumpt and Soulsby[101,121] for arthropods are followed. Taxonomy is not of great importance to the clinician except to know that closely related species tend to share similar life cycles, which aids in more effective management planning. For instance, if a particular parasite is known to be a strongyle, plans to control the parasitism may be based on knowledge of the probable life cycle and susceptibility to certain anthelmintics. Parasitism in one form or another has existed for hundreds of millions of years. It is part of the ecologic system of animals. In a pristine natural environment, hosts and their parasites often establish an equilibrium and coexist.

The presence of mature helminths or their eggs (ova) or larvae in the feces of an elephant, or finding them at necropsy, is not unequivocal proof that they are responsible for the animal's illness. A healthy wild animal may harbor large numbers of protozoal and helminth parasites without showing clinical signs of disease. Parasitic disease results when the equilibrium between parasite and host is upset, as the following situation illustrates. A healthy zebra *Equus burchelli* in Kruger National Park in South Africa was collected as part of an ongoing study to determine the seasonal parasite load of animals in the park. At necropsy, a thorough search was made of organs and tissues, and over 30 million individual parasites were counted or estimated. Furthermore, 14 different parasitic genera were represented. If that zebra had been subjected to malnutrition or other environmental stresses, however, clinical parasitism could easily have ensued.

Every attempt should be made to identify parasites obtained from elephants. Submit parasites to a veterinary parasitologist in the country where the elephant resides. Moving specimens across country borders may be a problem in many countries.

In North America, if possible, collect and submit specimens of helminths to the National Helminthological Museum in Washington, DC. This will help provide a better collective resource for elephant parasites.

## EVOLUTION OF HOSTS

The evolution of most animal species is not precisely known. However, a few species, such as the elephant and horse, have left a good paleontologic trail and an understanding of their evolution may help to illustrate some principles.

Elephants originated in Africa in the late Eocene epoch. Elephantine ancestors no larger than a pig gradually spread to all the northern continents. Numerous species developed and then became extinct. The height of elephant evolution occurred in the Pleistocene epoch. All species displayed varying degrees of a large flexible proboscis and one or more pairs of incisors that became massive tusks. The Asian elephant *Elephas maximus*, African bush elephant *Loxodonta africana,* and African forest elephant *Loxodonta africana cyclotis* are the only species that survived into the present epoch.

The parasitic fauna of prehistoric proboscideans is unknown, but it is likely that parasitic species similar to present-day elephant parasites infested ancient elephants.[42] Only recently have studies been conducted to indicate parasite burdens in current free-ranging and captive elephants. Recently captured Asian elephants

**Table 12.1.** Parasites Reported from Asian Elephants *Elephas maximus*

**Arthropods**
Class Insecta
  Order Mallophaga—Biting lice
    Suborder Rhynchophthirina
      *Haematomyzus elephantis*
  Order Siphunculata (Anoplura). Sucking lice
    No sucking lice parasitize elephants
  Order Siphonaptera. Fleas
      *Vermipsylla* sp.
  Order Diptera. Flies
    Suborder Nematocera
      Family Culicidae. Mosquitoes
      Family Simuliidae. Black fly, gnats
      Family Ceratopogonidae. Biting midges, "no-see-ums"
    Suborder Brachycera
      Family Tabanidae
        *Tabanus* sp. Horse fly, deer fly
      Family Muscidae
        *Musca domestica.* House fly
        *Musca autumnalis.* Face fly
        *Stomoxys calcitrans*. Biting stable fly
        *Glossina* spp. Tsetse flies
      Family Calliphoridae. Blow flies, bottle flies
        *Cochliomyia hominivorax.* Primary screwworm
        *Chrysomya bezziana.* Old World screwworm
        *Elephantoloemus indicus.* Asian elephant skin maggot
      Family Gasterophilidae
        *Cobboldia elephantis.* Asian elephant stomach bot fly
Class Arachnida
  Order Acarina
    Suborder Metastigmata. Ticks
      Family Ixodidiae. hard-bodied ticks, various genera
**Protozoa**
Subphylum Sarcomastigophora. Flagellates
  *Trypanosoma evansi*
Subphylum Apicomplexa (Sporozoa)
  *Toxoplasma gondii*
**Helminths**
Class Trematoda. Flukes
  *Fasciola hepatica*
  *Fasciola jacksoni*
  *Protofascia robusta*
  *Pseudodiscus collinsi*
  *Pseudodiscus hawkesii*
  *Pfenderius papillatus*
  *P. birmanicus*
  *P. heterocaeca*
  *Gastrodiscus secundus*
  *Bivitellobilharzia nairi*
Class Eucestoda. Tapeworms
  Family Anoplocephalidae
    *Anoplocephala manubiata*
**Nematodes**
  Order Rhabditida
    Family Strongyloididae
      *Strongyloides elephantis*
Order Strongylidea
  Family Strongylidae
    Chonianguin
      *C. epistomum*
      *C. magnostomum*
    Equinubria
      *E. spunculiformis*
    Decrusia
      *D. additictia*
      *D. decrusi*
  Family Cyanthostomidae
    Murshidia
      *M. murshida*
      *M. elaphasi*
      *M. falcifera*
      *M. indica*
      *M. lanei*
    Quilonia
      *Q. renniei*
      *Q. travencra*
    Khalilia
      *K. pileata*
  Family Ancylostomidae
    Bunostomum
      *B. foliatum*
    Bathmostumum
      *B. saneri*
    Grammocephalus
      *G. varedatus*
      *G. hybridatus*
  Family Syngamidae
    Mammomonogamus
      *M. indicus*
  Family Atractidae
    Leiperenia
      *L. galebi*
Order Ascaridided
  Family Ascaridae
    Toxocara
      *T. elephantis*
Order Spiruridea
  Family Acuaridae
    Parabronema
      *P. indicum*
      *P. smithi*
Order Filariidea
  Family Dipetalonematidae
    Indofilaria
      *I. pattabiramani*
    Dipetalonema (Loxodondofilaria)
      *D. asiatica*
  Family Setariidae
    *Stephanofilaria assamensis*

may be severely affected by intestinal flukes. The stress of captivity and transport may lead to overt disease.

Numerous reports of parasites in elephants are found in the literature. A bibliography of elephant parasites was published in 1922.[2] Many of these discuss therapy without precise identification of the parasite.[24,29,68,85] Table 12.1 lists the parasites that have been found in Asian elephants. Table 12.2 lists the parasites that have been found in African elephants. Note that all elephants harbor the same genera of parasites, but species usually differ. Name changes have been common over the years, so correlating older literature with modern nomencla-

**Table 12.2.** Parasites Reported from African Elephants Loxodonta africanus

**Arthropods**
Class Insecta
- Order Mallophaga. Biting lice
  - Suborder Rhynchophthirina
    - *Haematomyzus elephantis* Elephant louse
- Order Siphunculata (Anoplura). Sucking lice
  - No sucking lice on elephants
- Order Siphonaptera. Fleas
  - *Vermipsylla* sp.
- Order Diptera. Flies
  - Suborder Nematocera
    - Family Culicidae. Mosquitoes,
    - Family Simuliidae. Black fly, gnats
    - Family Ceratopogonidae. Biting midges, "no-see-ums"
      - *Culicoides toroensis*
      - *C. kanagai*
      - *C. loxodontis*
  - Suborder Brachycera
    - Family Tabanidae
      - *Tabanus* sp. Horse fly, deer fly
  - Suborder Cyclorrhapha
    - Family Muscidae
      - *Musca domestica.* House fly
      - *Musca autumnalis.* Face fly
      - *Stomoxys calcitrans*. Biting stable fly
      - *Glossina* spp. Tsetse flies
        - *G. pallipides*
        - *G. longipenis*
    - Family Calliphoridae. Blow (bottle) flies
      - *Cochliomyia hominivorax*. Primary screwworm
      - *Chrysomya bezziana*. Old World screwworm
    - Family Oestridae. Bot flies
      - *Pharyngolobus africanus.* African elephant throat bot fly
    - Family Gastrophilidae
      - Platycobboldia loxodontis. Blue elephant stomach bot fly
      - *Rhodhainomya roverei*. Green elephant stomach bot fly
      - *Ruttenia loxodontis*. African elephant skin maggot
      - *Neocuterebra squamosa*. African elephant foot fly

Class Arachnida
- Order Acarina
  - Suborder Metastigmata. Ticks
    - Family Ixodidiae. Hard-bodied ticks
      - *Amblyoma tholloni*
      - *Dermacenter cirumguttatus*
      - Various other genera and species
  - Suborder Mesostigmata. Mites
    - *Loxanoetus bassoni.* Small ear mite

**Protozoa**
- Subphylum Sarcomastigophora. Flagellates
  - *Trypanosoma congolense*
  - *T. brucei*
- Subphylum Apicomplexa (Sporozoa)
  - *Toxoplasma gondii*
  - *Cryptosporidium parvum*

**Helminths**
Class Trematoda. Flukes
- *Fasciola hepatica*
- *Fasciola jacksoni*
- *Bivitellobilharzia loxodontae*
- *Brumptia bicaudata*
- *Protofasciola robusta*
- *Eurytrema pancreaticum*
- *Dicrocoelium dendriticum*

Class Eucestoda. Tapeworms
- Family Anoplocephalidae
  - *Anoplocephala mpwapwae*

**Nematodes**
- Order Strongylidea
  - Family Strongylidae
    - Chonianguin
      - *C. algericum*
    - Equinubria
    - Decrusia
  - Family Cyanthostomidae
    - Murshidia
      - *M. linstour*
      - *M. hadia*
      - *M. longicaudata*
      - *M. brachyscelis*
      - *M. africana*
      - *M. anisa*
      - *M. dawoodi*
      - *M. omoensis*
      - *M. brevicapulatus*
      - *M. memphisia*
      - *M. aziza*
      - *M. loxodontae*
      - *M. soundanensis*
      - *M. brevicaudata*
      - *M. neveu-lemairei*
      - *M. vuylstekae* (cyclotis)
      - *M. witenbergi* (cyclotis)
    - Quilonia
      - *Q. apiensis*
      - *Q. africana*
      - *Q. Uganda*
      - *Q. brevicauda*
      - *Q. ethiopica*
      - *Q. khalili*
      - *Q. loxodontae*
      - *Q. magna*
    - Khalilia
      - *K. sameera*
  - Family Ancylostomidae (hookworms)
    - Bunostomum
      - *B. brevispiculum*
      - *B. hamatum*
    - Gammocephalus
      - *G. clathrates*
      - *G. intermedius*
  - Family Syngamidae
    - Mammomonogamus
      - *M. loxodontus*
  - Family Atractidae
    - Leiperenia
      - *L. leiperi*
      - *L. morelli*
- Order Spiruridea
  - Family Acuaridae
    - Parabronema
      - *P. africanum*
      - *P. rhodesiense*
      - *P. longispiculatum*
- Order Filariidea
  - Family Dipetalomeatidae
    - Indofilaria
      - *I. pattabiramani*
    - Dipetalonema (Loxodontofilaria)
      - *D. gossi*
      - *D. loxodonti*
- Order Enoplida
  - Superfamily Trichinelloidea
    - *Trichuris* sp. Whipworm

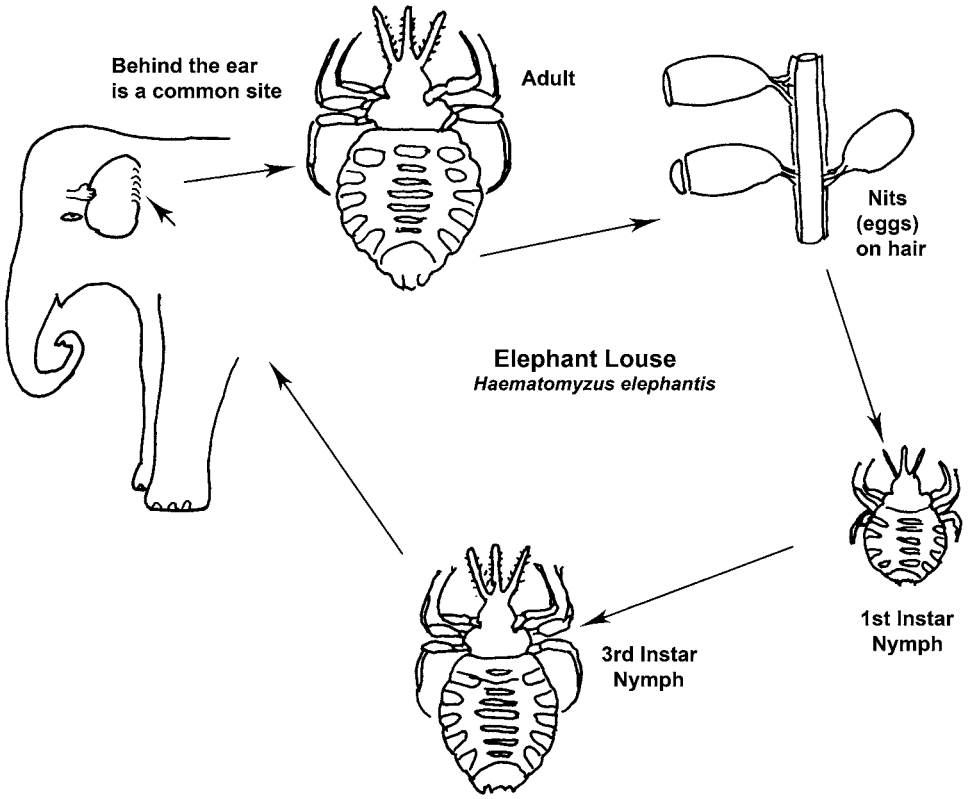


***Figure 12.1.*** *Life cycle of the elephant louse* Haematomyzus elephantis.

ture is difficult.[33,86] Those parasites that have been associated with clinical disease in elephants will be discussed in more detail.

## EXTERNAL PARASITES

### Lice

Neither Asian nor African elephants are parasitized by sucking lice.[36,41] Both species harbor a biting louse, *Haematomyzus elephantis*.[55,67,73,88]

**Identification.** Lice are wingless insects that live a complete life cycle on a single host. They are generally host specific, i.e., lice found on livestock and pets will not spread to elephants or vice versa. The elephant louse is large (3 mm long) (see Table 12.5) and easily seen with the unaided eye.[106]

Taxonomically, elephant lice are in the order Mallophaga (biting lice); however, biologically they are blood-sucking lice. They attach to the skin through an elongated proboscis with mouth parts at the tip.[41]

**Life cycle.** The life cycle of lice is simple (Fig. 12.1). Adult lice copulate and the female deposits fertilized eggs (nits) on hairs and cements them in place. The eggs hatch within 1 to 3 weeks and a tiny nymph, a replica of the adult louse, emerges. As the louse matures it undergoes two or three molts, during which it increases in size but does not change body structure. The development to maturity requires 1 to 2 weeks. The entire life cycle may be completed in as little as 2 to 5 weeks. Adults live for 15 to 40 days.

**Epizootiology.** Lice may complete their life cycle on a single animal. There is no free-living stage. Transmission from one elephant to another is by close body contact, as may occur with maternal care of infants, during breeding, or when individuals touch each other during greetings or other social interactions. Populations of lice tend to build up during the colder months of the year. Although the cycle may be completed in as little as 2 to 5 weeks, it may be extended many months by arrested development in the nit stage, or a few lice may lie dormant until environmental conditions become conducive to rapid development of a population. Lice are most common in captive elephants,[8,56] but they may also be seen in free-ranging elephants.[14]

**Clinical signs.** Dermatitis may or may not be present, but with heavy infestations, pruritus may be pronounced, evidenced by frequent scratching, which may traumatize the skin.[59] Damage to the cornea may result from scratching (see Chapter 35, Figure 35.16). Some affected elephants are restless or hyperirritable and lash the tail against objects or sides of the body.[23,98] The areas of the body most likely to be infested are behind the ears and around the base of the tail, although in severe infestation lice have been seen over much of the

body surfaces, especially in folds of the skin in the axillary region or other hidden areas.[56,57,108] The skin may be dry and scaly. The egg cases (nits) may be observed attached to hairs (Fig. 12.1).[81]

**Management.** Lice may or may not be destroyed by direct contact with numerous insecticides, including pyrethrins, chlorinated hydrocarbons, carbamates, and organic phosphates (see Table 12.6). Ivermectin at a dosage of 0.059–0.087 mg/kg administered subcutaneously has been effective.[57] Therapy must be repeated to allow eggs to hatch, because nymphs inside the egg are not killed by the initial treatment. Injectable ivermectin administered orally at a dose of 0.1 mg/kg is also effective (Mikota, unpublished data).

## Fleas

**Identification.** Fleas are wingless insects with laterally compressed bodies.[13] They vary in size from 1.5–4 mm. Fleas are not host specific; thus several different types may be found on elephants.

**Life cycle.** Adult fleas copulate and the female lays approximately 20 eggs at a time in detritus on the host or in dust/dirt in the elephant's environment. Larvae hatch in 2 to 16 days and feed on dried blood, feces, or other organic matter. The larval stage is completed in 7 to 10 days and the insect enters the pupal stage, which may last for 10 days or remain dormant for months. The adult emerges from the pupal case to seek a suitable host.

**Epizootiology.** Fleas may be transmitted from one elephant to another by close body contact. Fleas are active and may jump prodigious distances. Contamination of the captive environment with flea eggs and pupae enables infestation to be acquired by simply walking through any area previously exposed to animals with fleas. The life span of an individual flea may be over 1 year. During this time the female may lay as many as 400 eggs.

Fleas are notorious vectors for infectious and parasitic agents; however, no such instances have been documented in elephants.

**Clinical signs.** Adult fleas consume only blood and may cause anemia. While biting the host, fleas deposit saliva, which may stimulate an allergic response varying from mild irritation to marked pruritus, hyperemia, swelling, and dermatitis.

**Management.** Management for fleas is the same as for lice.

## Mosquitoes

**Identification.** Mosquitoes are slender-bodied, long-legged, winged insects with spherical heads and a long slender proboscis, part of which is the fascicle, used to cannulate venules to ingest blood meals from the host. Even though the elephant skin is thick, the bulk of the skin layer is made up of dermis. A mosquito may easily penetrate the epidermis and access the vascular bed, especially in the ears. The numerous species differ in habitat preference but have no significant host specificity. Elephants in mosquito territory will likely be bitten.

**Life cycle.** The gravid female lays eggs in water. The eggs hatch in less than a week to become air-breathing larvae. Larvae undergo four molts over a period of 2 weeks, progressing to the pupal stage that lasts 2 to 7 days. The adult emerges from the puparium and must dry itself before being able to fly off, usually within 24 hours. The entire cycle requires approximately a month, less in warm moist habitats.

**Epizootiology.** Because mosquitoes fly, they can easily reach hosts. They do not remain on the host but, rather, alight, ingest a blood meal, and leave. Like fleas, mosquitoes are vectors for numerous infectious diseases, serving as the intermediate host for some and as mechanical vectors for others.

**Clinical signs.** A swarm of mosquitoes may be annoying, but even in large numbers, will not likely cause anemia.

**Management.** Reducing mosquito numbers requires management of the environment (decreasing breeding habitat, insecticide treatment).

## Black Flies (Family Simuliidae; Buffalo Gnats)

**Identification.** Simulid flies are closely related to mosquitoes but have a short, piercing proboscis and a hump over the thorax. The legs are not as long as those of mosquitoes.

**Life cycle.** The female black fly lays eggs only in running water, either on the surface or on submerged stones, twigs, or vegetation. The eggs may remain in the water for months, overwintering in this state, or hatch in a few days. Larvae are mobile, with the loping type of ambulation typical of an "inchworm." Larvae molt six times before entering the pupal stage, which floats near the surface so that a special respiratory tube can take in the necessary air. Adults emerge from the puparium and may swarm in dense clouds if the population is large.

**Epizootiology.** Black flies are found throughout the world but are concentrated in warmer climates. Flies are active in the morning and evening. They do not live on hosts but take blood meals from them.

**Clinical signs.** Swarms of black flies are annoying to elephants and may inhibit feeding.

**Management.** Management for black flies is the same as for mosquitoes.

## Midges (Family Ceratopogonidae)

**Identification.** Five species of *Culicoides* spp. midges have been collected from African elephants: *Culicoides toroensis, C. kangai, C. loxodontis,* and two unidentified species.[70,71]

**Life cycle.** Adult midges are generally thought to be nocturnal, but those that feed on elephants appear to be diurnal. Midges feed on the area behind the ears where the epidermis is thinnest and smoothest. A common site is the upper border of the ear, which is constantly curved caudally, providing a permanent shady and moist spot.

The ears of the African elephant provide 20% of the animal's skin surface, and each ear weighs as much as 20 kg. Because the ear is a primary thermoregulatory organ, it is well supplied with a vascular network of capillaries.

The female midge takes a blood meal and leaves the elephant, retreating to an unknown location in vegetation to await maturation of her ovaries. The precise stimulus for returning to the host is unknown, but she reattaches to an elephant and takes another blood meal.

The gravid female is attracted to and oviposits in elephant dung. She may then return to an elephant to repeat the process. The eggs hatch and develop into adult midges.[70,71]

## Tabanids (Family Tabanidae; Horseflies, Deerflies)

**Identification.** Tabanids are medium to large flies with powerful wings and large eyes.

**Life cycle.** Females lay eggs in damp soil or decaying organic matter. The eggs are glued in masses near a water source so that larvae, which hatch in about 1 week, will fall into the water to continue maturation. Larvae burrow into the mud at the bottom of a pond or stream and subsequent pupae may overwinter there. Adults emerge from the puparium and require a prompt blood meal to complete the life cycle, which requires a minimum of 4 months or may extend to the next season.

**Epizootiology.** Tabanids are diurnal and are especially active on hot, bright, humid days. Only the females take blood meals every 3 or 4 days. The bite from a tabanid is painful. Tabanids may be responsible for transmission of parasites and other infectious agents, but this has not been documented for elephants. Tabanids are an important intermediate host for *Trypanosoma evansii*, the cause of trypanosomiasis (surra) in elephants and other artiodactylids in Africa and Asia.

**Clinical signs.** The large mouth parts may disrupt capillaries, which may continue to weep after the tabanid has departed, attracting other nonbiting flies. Such flies may annoy the elephant.

**Management.** No treatment or prevention is available other than swatting the fly if observed near or on the elephant. It is not uncommon for elephants to use branches as a tool to swat flies or brush them away.[51]

## Miscellaneous Flies (Family Muscidae)

**Identification.** Elephants are plagued with the same types of flies that afflict domestic livestock.[121] The housefly, *Musca domestica*, has four dark stripes on the thorax and yellow spots on the side of the abdomen and is approximately 7 mm long. The biting stable fly, *Stomoxys calcitrans*, is also annoying to elephants. It is the same size as the housefly but has a long, stiff proboscis and gray-brown spots on the abdomen. Other species of flies may annoy or afflict elephants. The species that are present will depend on the environment.

The tsetse fly, *Glossina* spp., is a biting fly that is responsible for transmitting the trypanosomes of sleeping sickness in Africa (a disease of humans). Two species, *Glossina pallidipes* and *G. longipenis,* are known to take blood meals from elephants.[100] Elephant blood was identified in tsetse flies collected in the Nguruman district of Kenya. The prevalence for elephant blood was 23.2% in the flies collected.[93]

**Life cycles.** Housefly females lay eggs on manure or decaying organic matter. The larvae hatch in less than 24 hours, grow, and molt twice in a few days to become third-stage larvae, which move to a drier area and pupate. Adults emerge in 2 to 3 weeks, climb to the surface, and spread their wings to dry. Adults live for 6 to 8 weeks, during which time a female will lay 2000 eggs. The entire cycle is completed in 3 to 5 weeks.

**Epizootiology.** Houseflies are found both inside and outside barns and sheds. All flies are more active on warm days than on cool ones and cease activity at dark.

**Clinical signs.** Flies are annoying to elephants. Biting flies may cause additional irritation. Flies are particularly irritating if there is excessive lacrimal secretion. They may exacerbate the problem, resulting in conjunctivitis.

**Management.** Fly control is a never-ending challenge to managers of any animal enterprise. Having access to dust baths or mud wallows is important for elephants to minimize fly annoyance. All the methods employed by livestock raisers have application with elephants. It is impossible to control flies in free-ranging elephants, but this is not as likely to be a problem because animals may choose to move away or seek the respite of water or a mud bath.

## Blowflies (Family Calliphoridae; Bottle Flies)

Numerous species of flies deposit their eggs in decaying flesh or in the feces of wild and/or domestic animals.[30,47] A few species invade fresh or nonnecrotic wounds. Space does not permit discussion of each of the possible species that may parasitize elephants (see Table 12.5). A discussion of a few representative species follows.

### *Chrysomya bezziana* (Old World screwworm)

Identification. The adult *Chrysomya bezziana* has a metallic green or blue body color. The legs are black or dark brown. Body length varies between 8 and 12 mm. The eggs are approximately 1.25 mm in length. The first instar measures up to 3 mm, the second instar up to 4–9 mm, and the third instar up to 18 mm.

The primary screwworm, *Cochliomia hominivorax*, is a New World fly, which has been eliminated from North America at the present time.

Life cycles. The life cycles of all screwworms are similar (Fig. 12.2). The female deposits fertilized eggs in batches of 150–500 on the margins of fresh wounds. Wounds as small as tick bites may be selected. The eggs hatch in 18–24 hours and the first instar larvae feed on blood or serum exuding from the wound.[119]

The second instar invades living tissue, and the third instar is buried in living tissue so that only the posterior end protrudes from the wound. After 3 or 4 days the maggot releases and falls to the ground for pupation, which varies from 7 to 9 days in duration (Fig. 12.2). Under favorable conditions, eight or more generations may develop each year. This fly also parasitizes human wounds and a broad range of mammalian hosts.

Clinical signs. Maggots are observed in the wound, with only the posterior peritremes visible, but these are characteristic of the species (Fig. 12.2).

Management. Management in free-ranging elephants is virtually impossible. In captivity, the wound should be cleansed and debrided to remove necrotic tissue. Larvae may be removed mechanically or killed by the instillation of insecticides into the wound. Because the vehicle used to solubilize the insecticide may be irritating to the tissue and will be absorbed into the system much more readily than through healthy skin, only as much fluid as is absolutely necessary to destroy the larvae should be applied. Chloroform, ether, and hydrogen peroxide will also cause the larvae to retreat from crevices and cavities. After larvae are destroyed, the wound should be properly treated and dressed. The wound should be monitored to remove missed larvae or deal with reinvasion. Ivermectin is also effective.

**Bot flies (family Oestridae).** The only osterid bot fly found associated with the elephant is *Pharnygolobus africanus*, the African elephant throat bot fly (see Table 12.5). The third instar larvae of this fly are located in the pharynx.

**Bot flies (family Gastrophilidae).** See Table 12.5 for a listing and biological information about these species. A description of two species follows.

### *Ruttenia loxodontis*

Biology. Two elephant calves imported into Czechoslovakia had white specks on the skin of the abdominal flanks and thighs. The lesions caused no discomfort and

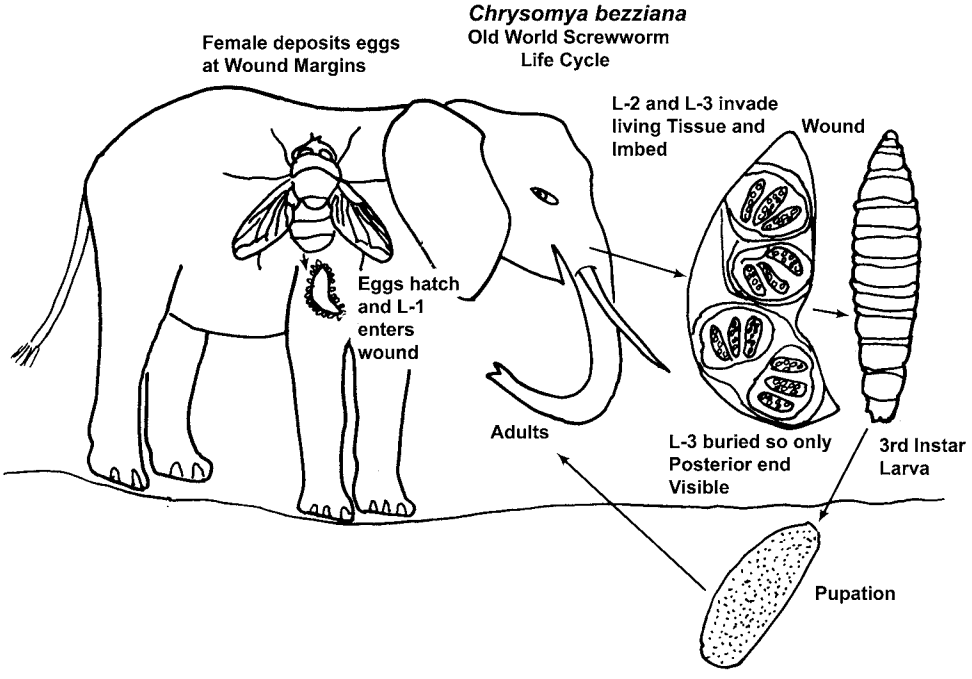


***Figure 12.2.*** *Life cycle of the Old World screwworm* Chrysomya bezziana.

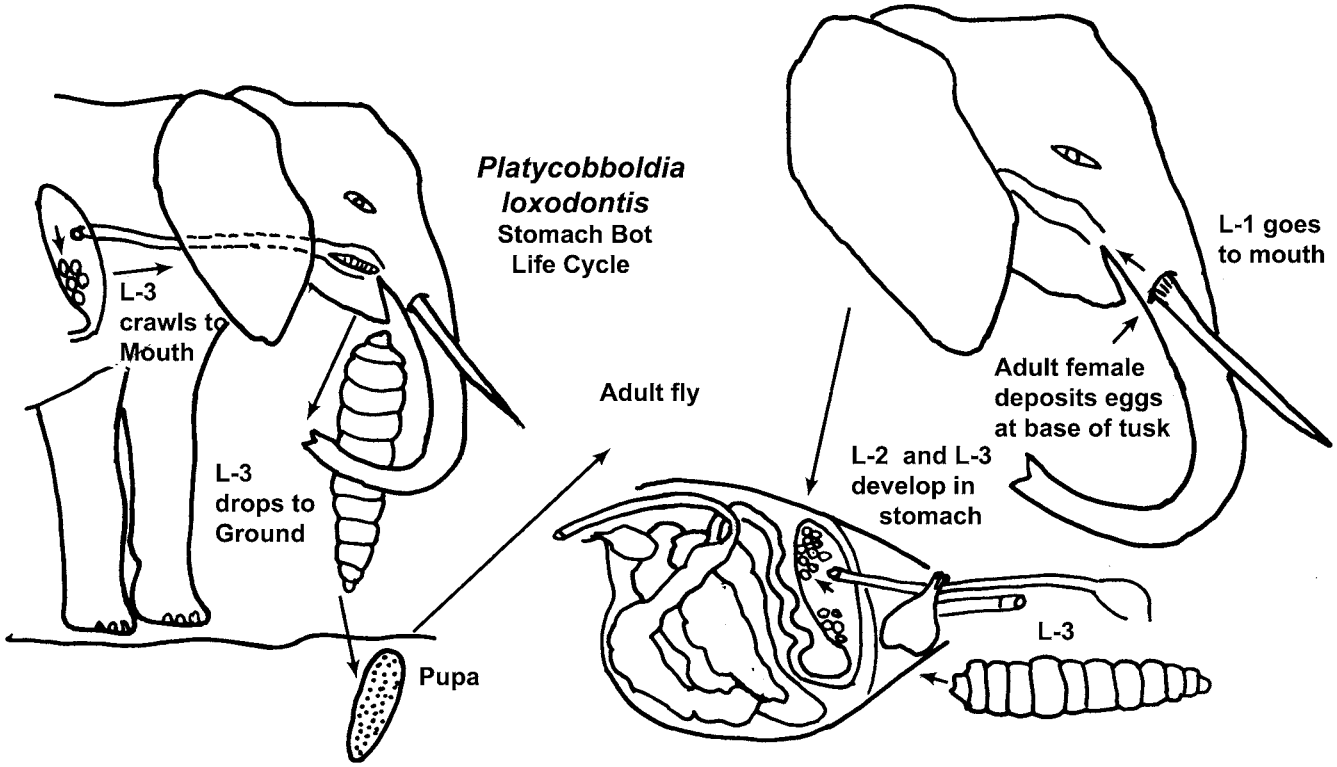


***Figure 12.3.*** *Life cycle of the African elephant stomach bot fly* Platycobboldia loxodontis.

swelling was not observed. Four months after arrival, the calves were moved to a heated barn, 3 months later the lesions swelled, and in another month the swellings ruptured, exposing third instar larvae. The life cycle of this parasite is unknown.[102]

### Stomach bots

Identification. *Platycobboldia loxodontis*, the blue elephant stomach bot fly, and *Rodhainommia roverei,* the green elephant stomach bot fly, parasitize the African elephant. *Cobboldia elephantis,* another stomach bot, parasitizes Asian elephants.[63] See Figure 35.15 in Chapter 35. See Table 12.5 for biological data.[39,66,120]

Life cycles. The life cycle of elephant stomach bots is similar to that of *Gastrophilus* spp., found in horses and rhinoceroses (Fig. 12.3); however, the eggs are deposited at the base of the tusk in elephants.

A hunter in India found the bases of the tusks packed with kilograms of unknown dipteran larvae.[34] It is a commonly held belief that the third instar larvae are passed in the feces, but modern investigators have determined that they usually crawl up the esophagus and exit the body via the mouth. Larvae may be seen in the feces following deworming. Ulceration and possible gastric rupture may occur in heavy infestations.

Clinical signs. Moderate infestations of the third instar larvae in the stomach of elephants cause little harm and no demonstrable clinical signs. Heavy infestations may cause gastritis and general signs of gastrointestinal tract parasitism. This parasitism is not likely to be seen in captivity, except for newly imported elephants. After an animal is cleared of an existing infestation, the lack of suitable habitat precludes continuation of the cycle for the fly.

Diagnosis. Close observation may reveal adult flies on or in the vicinity of elephants. In free-ranging elephants, eggs and first instar larvae may be seen at the base of the tusk.[34,113] Third instar larvae may be observed at necropsy or in the enclosure of captive animals.

Management. Avermectins (ivermectin, moxidectin) are the anthelmintics of choice administered orally to rid the elephant of third instar larvae. See Table 12.6.

## Ticks

**Identification.** There are two major groups of ticks: hard-bodied ticks (family Ixodidae) and soft-bodied ticks (family Argasidae). No soft-bodied ticks parasitize elephants; however, there are numerous genera of hard-bodied ticks and it is likely that those that are not host specific may be identified according to the locality inhabited by elephants. Tick paralysis has not been reported in an elephant as a result of hard-bodied tick attachment.

The African elephant is the primary host for two ixo-

did ticks: *Amblyoma tholloni* and *Dermacentor circumguttatus*.[75] Other species that have been found on elephants include *Amblyoma asterion, A. cohaerens, A. gemma, A. nuttallii, A. paulopunctatum, A. sparsum, A. variegatum, Boophilus microplus*,[76] *Dermacentor rhinocerinus, Haemophysalis leachii, Rhipicephalus appendiculatus, R. compositus, R. humeralis, R. longus, R. maculatus, R. muehlensi, R. parvus, R. pulchellus, R. snegalensis, and R. simus.* Domestic livestock or other wild animals are primary hosts for the latter species.

*Amblyoma tholloni* is known to transmit *Cowdria ruminantium*.[75]

### Mites

None of the common genera of mites found on domestic and wild animals (Sarcoptes, Psoroptes, Chorioptes) have been reported from elephants. A small ear mite, *Loxanoetus bassoni,* has been found on African elephants.[35,40]

## INTERNAL PARASITES

### Protozoa

Protozoal diseases are important throughout the world; however, elephants have a meager protozoan fauna.

#### *Toxoplasma gondii.*

Biology. Little is known about toxoplasmosis in elephants. In a study in Thailand, blood samples were collected from 156 Asian elephants over the course of 5 years. Samples were tested for antibodies to *Toxoplasma gondii* by both a modified agglutination test and a latex agglutination test. The prevalence of positive antibody titers varied from 25.6–45.5%.[109] Titers to toxoplasmosis (up to 1:400) were also found in 14 of 45 (32%) clinically healthy elephants in Sri Lanka.[32] Such findings indicate exposure and response to the antigen, but not necessarily disease.

Some studies in Africa did not demonstrate antibody titers to Toxoplasma,[96] but others did.[53,87] No clinical disease or pathology in elephants has been ascribed to toxoplasmosis.

### Trypanosomiasis (Surra)

**Identification.** *Trypanosoma evansi* is the only trypanosome affecting Asian elephants, causing a disease called *surra*. Surra is an Indian word meaning "rotten." The African elephant has been reported to be infected with *T. congolense* in Tanzania and Mozambique and *T. brucei* in Uganda.[16,52]

Trypanosome species are similar, and species identification may be impossible without animal inoculation and other sophisticated diagnostic procedures. They have a leaflike shape, with a single flagellum attached to the cell by a undulating membrane.

**Life cycle.** *T. evansi* does not require a period of maturation in an insect vector and thus has a noncyclic transmission. Any biting or blood-sucking insect or tick may serve as a vector. Flies of the genera Tabanus and Stomoxys are commonly implicated. Mechanical transmission by contaminated hypodermic needles is also possible.

**Epizootiology.** The geographic distribution of *T. evansi* is extensive, occurring in North Africa, Asia Minor, countries of the former U.S.S.R., Pakistan, Afghanistan, India, Burma, Malaya, Indochina, South China, Indonesia, and the Philippines.[16,74,106]

**Clinical signs.** Acute surra is characterized by fever, depression, weakness, and edema. The presence of pulmonary edema may contribute to the development of secondary pneumonia. Females may abort, and the milk of lactating females may become caseous. Large numbers of trypanosomes are seen in peripheral blood samples. Death may occur within a few weeks.[26]

Chronic surra is characterized by intermittent episodes of fever, anemia, dependant edema, and emaciation. Between episodes of fever, the parasite may be absent from peripheral blood vessels. An elephant may live for 3 or 4 years, depending on the care provided.

**Diagnosis.** Trypanasomes may be difficult to detect in peripheral blood. Dozens of laboratory tests have been used with less than resounding success. PCR technology is perhaps the brightest hope for diagnosis

**Management.** Many drugs have been used in an attempt to eliminate trypanosomes from various species of animals. Host responses to the drugs have been variable and unreliable. Drugs that have been used in India include melarsomine (Cymelarsan) and suramin (naganol).

### Coccidiosis

Coccidiosis is common in most ungulate species. Surprisingly, no *Eimeria* spp. or *Isospora* spp. have been reported from either the Asian or African elephant.[92] *Cryptosporidium parvum* has been reported only from the Barcelona, Spain Zoo.[45,48,64]

**Clinical signs.** No clinical signs of cryptosporidiosis have been reported in elephants. In some species the organisms invade the epithelial mucosa of the small intestine, causing enteritis and diarrhea.

***Babesia* spp.** *Babesia* spp. have been found in African elephant blood.[15,96] Another piroplasm *Nuttallia loxodontis* has been described.[96]

It may be important to mention here that other blood parasites have been identified in elephants, including microfilarial larvae (White 1980).[115]

### Intestinal Ciliates

Numerous species of ciliate protozoa have been isolated from ingesta in the intestines, including 11 species of

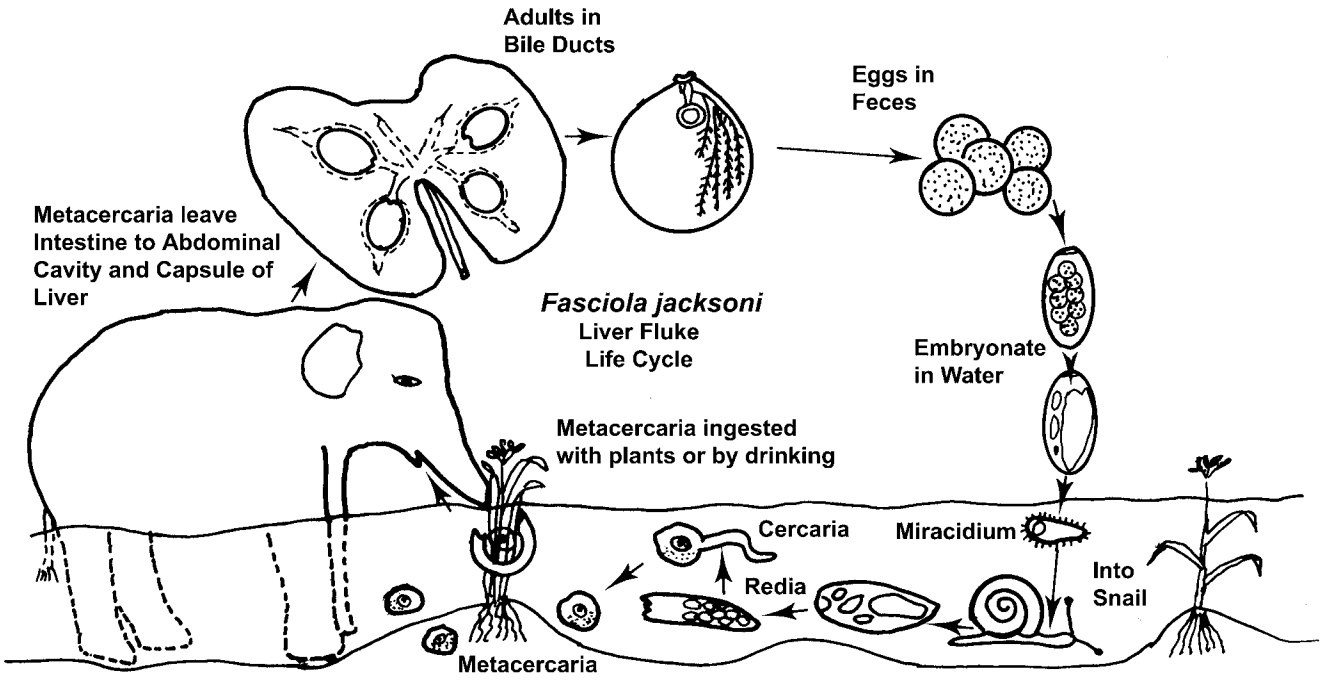


***Figure 12.4.*** *Life cycle of the elephant liver fluke* Fasciola jacksoni.

the genus *Triplumaria. T. acuticaudata and T. nucleocaudata* were identified in *Loxodonta africanus*, and the other 9 species—*T. hamertonii, T. selenica, T. longinucleata, T. heterfasciculata, T. antis, T. doliiformis, T. irregularis, T. poljanskii,* and *T. ovina*—were found in *Elephas maximus*.[104,105] It is currently thought that these protozoa aid in the utilization of cellulose by elephants. It also appears that many of the species are unique to elephants.[38,72,99] None are known to be pathogenic.

## Trematodes (Amphiostomes, Paramphiostomes)

**Identification.** *Fasciola jacksoni* is a liver fluke unique to elephants. See Table 12.3 for details on biology.[17,43,54,01,07,116] The common liver fluke, *Fasciola hepatica* has also been found in elephants and is locally ubiquitous and found throughout tropical and temperate regions of the world.

**Life cycle.** The life cycles of both species are similar (Fig. 12.4).

Adult flukes live in the bile ducts. Some may be found in the lung. Eggs are discharged into the bile duct, carried to the intestine, and excreted in the feces. The eggs must fall into water for maturation to the ciliated miracidium stage. This requires 10 to 12 days. The miracidium bores into one of many species of amphibious snails. *Lymnaea truncatula* is one of the more common intermediate hosts, but other species of Lymnaea may also act as hosts. While in the snail, the miracidium loses its cilia and matures to become a sporocyst, and then a redia, and finally a cercaria. This phase requires 4.5 to 7.0 weeks.

Cercaria leave the snail and are free swimming for a few minutes to 2 hours. They attach to a plant just below surface level, lose their tail, and become metacercaria, which is the infective stage for elephants. Metacercaria may remain suspended in the water, but most will sink to the bottom of the body of water. Metacercaria are ingested with forage or by drinking. Immature flukes (marita) are released into the duodenum, where they penetrate the wall of the intestine, enter the peritoneal cavity, and migrate to the liver. By 3 to 7 days after infection, the majority of the young flukes will have reached the liver, where they penetrate the capsule. A migratory period of some 5 to 6 weeks in the liver parenchyma ensues before the flukes enter the bile duct and mature.

The prepatent period is about 8 weeks, but development may be retarded, delaying maturity another 2 months. Adult flukes may live for 9 months or longer.

**Epizootiology.** The completion of the fluke life cycle is dependent on the presence of snail intermediate hosts, which, in turn, are dependent upon an aquatic environment. The eggs will not develop at temperatures below 10°C, and there is a direct correlation of development time with temperature (at 13°C [55.4°F], 60 days; at 15°C [59.0°F], 40 days; and at 26°C [78.8°F], 12 days).

Some snail hosts are capable of estivation for as long as 13 months under certain circumstances, e.g., during

**Table 12.3.** Biological Data for Selected Asian Elephant Parasites

| Scientific Name | Common Name | Adult _ mm | Adult _ mm | Eggs µ | Intermediate Host | Location, Adult |
|---|---|---|---|---|---|---|
| **Nematodes** | | | | | | |
| *Murshidia* | GI nematode | 24.0–28.2 | 20.0–23.0 | 72 × 48 | None | Large intestine, cecum |
| *Murshida* | | | | | | |
| *Murshidia falcifera* | Same | 29.0–35.6 | 22.0–28.8 | 84 × 41 | None | Large intestine |
| *Murshidia indica* | Same | 21.2 | 16.7 | 56 × 29 | None | Stomach, large intestine |
| *Quilonia renniei* | Same | 20.0 | 15.0 | 75 × 37 | None | Cecum |
| *Quilonia travancra* | Same | 20.0 | 18.0 | 70 × 45 | None | |
| *Amira pileata* | Same | 13.9–19.9 | 12.5 | 36 × 72 | None | Large intestine, cecum |
| *Choniangium epistemum* | Same | 18.5 | 14.0 | 50 × 25 | None | Cecum |
| *Equinubria sipunculiformis* | Same | 24.0–28.0 | 23.0–27.0 | 63 × 36 | None | Cecum |
| *Decrusia additicta* | Same | 14.0–17.0 | 15.0–20.0 | 80 × 36 | None | Large intestine, cecum |
| *Bunostomum foliatum* | Stomach hookworm | 15.2 | 13.0 | | None | Stomach |
| *Bathmostomum sangeri* | Intestinal hookworm | 18.0–22.1 | 15.0–16.6 | 63 × 33 | None | Large intestine |
| *Gramocephalus varedatus* | Bile duct hookworm | 47.0 | 55.0 | 68 × 37 | None | Bile ducts |
| *Syngamus galebri* | Pharyngeal gapeworm | 30.0 | 8.5 | 100 × 52 | None | Pharynx |
| *Toxocara longoptrera* | Ascarid | | | | None | Bile ducts |
| *Parabonema indicum* | Stomach worm | 13.0 | 9.0 | ? | None | Stomach |
| *Leiperenia galebi* | Intestinal nematode | 3.8 | 3.25 | ? | None | Intestine |
| *Strongyloides elephantis* | Rhabditid intesinal worm | 2.6–3.6 | None as parasites | 23 × 49 | None | Small intestine |
| **Trematodes** | | | | | | |
| *Pseudodiscus collinsi* | | 3.0–7.0 wide<br>5.0–12.0 long | Hermaphroditic | 135 × 86 | | Large intestine |
| *Pseudodiscus hawkesii* | | 2.0–3.0 wide<br>3.5–6.0 long | Hermaphroditic | 135 × 70 | | Large intestine |
| *Pfenderius papillatus* | | 2.5–2.75 wide<br>4.5–5.5 long | Hermaphroditic | 150 × 70 | | Large intestine |
| *Fasciola hepatica* | Liver fluke | 13.0 wide<br>30.0 long | Hermaphroditic | 140 × 80 | Amphibious snails—*Lymnaea* spp. | Bile ducts |
| *Fasciola jacksoni* | Liver fluke | 9.0–12.5 wide<br>12.0–14.0 long | Hermaphroditic | 115 × 58 | Amphibious snails | Bile ducts |
| **Cestodes** | | | | | | |
| *Anaplocephala manubriata* | Tapeworm | 2.6 cm long<br>1.6 cm wide<br>2.5–3.0 mm thick | Hermaphroditic | 50 × 55 | | Intestine |
| **Protozoa** | | | | | | |
| *Trypanosoma evansi* | Causes Surra | 15 to 34 µ long | | | None, mechanical vector, biting flies | Blood |

drought conditions. The infection may be maintained while the snail is buried in the dried mud.

Metacercaria may persist for a few days to a few weeks while encysted on the plant. Some of the cysts may fall to the bottom of the water and be stirred up and ingested when an animal walks into the water to graze or drink. The cyst may survive for as long as 8 months on moist hay, but the usual drying process will shorten life to a few weeks.

The prevalence of *F. jacksoni* in Asian elephants was studied in Assam, India.[90,91] Free-ranging elephants had a prevalence of 33.78%, and captive elephants showed prevalence of 18.18–62.28% depending on the location.

**Clinical signs.** Both acute and chronic forms of fascioliasis have been seen. The acute form occurs with overwhelming infections that produce signs of hepatic insufficiency, such as anorexia, constipation, diarrhea, anemia, icterus, and death similar to those caused by other agents.[90] The chronic form is more often seen. Chronic stasis of the bile, caused by flukes obstructing the ducts, produces a hepatic fibrosis, which ultimately causes an elevation of intrahepatic blood pressure. A hyperplastic cholangitis, which allows leakage of plasma protein causing hypoproteinemia, also occurs. Adult flukes suck blood, causing intrabiliary hemorrhage, which results in anemia. If the infestation involves the lungs, respiratory signs may be seen.

Elephants become anorectic, accompanied with weight loss. Mucous membranes may be pale or icteric, and pendant edema may be seen. Depression and emaciation follows anorexia. Either diarrhea or constipation may be seen. Signs are most severe in young elephants.[21,90]

**Diagnosis.** The eggs may be observed in fecal flotation. Elevated liver enzymes may be detected on blood chemistry analysis, as may elevated clearance times for BSP.[18] The clinical pathology associated with fascioliasis in four elephants in Myanmar included decreased hemoglobin 6.8–9.0 g/dl (normal 12.1), packed cell volume 19–28% (normal 34), erythrocytes 1.53–2.3 $\times$ $10^6$/µ (normal 3.18), plasma protein 5.4–8.6 g/dl (normal 9.0), and leukocytes 14.5–16.0 $\times$ $10^3$/mm$^3$ (normal 14.7). A bromosulfathalein (BSP) liver function test was performed with halftime clearance ranging from 4.3–6.5 minutes. Normals haven't been established.[18] The number of fluke eggs varied from 6–83/g of feces.

At necropsy, infected livers had hemorrhagic tracts, thickening of bile ducts, cirrhosis, and pseudolobulation.[21] The parasites were also found in the bronchi of the lung, causing bronchitis with desquamated cells and a fibrinohemorrhagic exudate.[97]

**Management.** Many different drugs have been used to treat fluke infestation. Currently, the only recommended drug in the United States is clorsulon (Curatrem®) at a dose of 7 mg/kg body weight. This is given per os, twice at 45–60-day intervals. Albendazole is also effective. In India, triclobendazole at 9 mg/kg (not to exceed 7200 mg/animal) and oxyclozanide 7.5 mg/kg (not to exceed 6.0 g/animal) were used successfully.[90]

### Other species of trematodes

Identification. Both immature and mature *Gastrodiscus secundus* and *Pseudodiscus collinsi* were recovered from the cecum of free-ranging Asian elephants.[7–9,91] *Pseudodiscus hawkesii* and *Pfenderius papillatus* were found in Sumatran elephants.[66] The blood fluke, *Bivitellobilharzia nairi,* has been recovered from Asian elephants in India.[85]

Life cycle. The precise life cycles of these trematodes are unknown.

Additional comments. The pathology associated with *Gastrodiscus secundus* and *Pseudodiscus collinsi* include petechia and ulcers of the cecal mucosa, with microscopic lesions being mild lymphocytic infiltrates with focal necrosis of villi.[91]

*Protofasciola robusta* may cause significant pathology. The walls of the colon are thickened and edematous. Numerous trematodes may be seen firmly attached to the wall, along with a hemorrhagic colitis. At the site of attachment of the fluke, it pulls up a segment of the mucosa into the sucker on the fluke. A pseudomembrane composed of necrotic debris may be formed in the vicinity of the attached flukes.[111]

Mebendazole was used in Thailand to treat *Pfenderius papillatus* infestation.[19,78]

## Cestodes (Tapeworms)

**Hydatid disease.** Although hydatid disease, caused by *Echinococcus granulosus*, has a worldwide distribution, reports of hydatid cysts in elephants are rare.[12,118] Perhaps this is because the elephant doesn't enter into any of the carnivore/herbivore cycles.

**Other tapeworms.** The tapeworm fauna of elephants is sparse. *Anoplocephala* sp. proglotids were identified in the feces of a 2-year-old Asian elephant that had a gastrointestinal impaction[112]. The author assumed she had acquired the tapeworm from an older cow that was known to be parasitized with anoplocephala.[112] *Anoplocephala manubrisyi* has been recovered from elephants.[114,117] Many tapeworms in other species cause little or no clinical disease, which is probably true for elephants as well.

**Identification.** The scolex has neither a rostellum nor hooks. Proglottides are wider than long. Eggs vary in size from 50–80µ and each egg has three outer membranes with a pyriform apparatus (pear-shaped with hooks on one side)[13].

**Life cycle.** The proglottides are bisexual, with fertilized eggs produced as the proglottid matures. The eggs pass out in the feces and are ingested by orobatid mites where they continue maturation to become a cysticercoid. Elephants acquire infection by ingesting forage containing the mites with the cysticercoid larvae. The embryo is released in the intestine to develop into the tapeworm.

**Clinical signs.** It is doubtful if tapeworms cause clinical disease in elephants.

**Diagnosis.** Frequently it is not possible to identify eggs in the feces. In horses an ELISA test is used to detect infection with Anoplocephala.[13]

## Nematodes (Phylum Nematohelminthes-Nematoda)

Nematodes are the most numerous and most detrimental of the elephant parasites. The taxonomic outline at the beginning of this chapter lists the nematodes that have been reported from elephants. Most of these adult parasites are located in the gastrointestinal tract (GI). Many aspects of GI parasitism are similar, regardless of which species of parasite is involved. The following introductory remarks obviate the need to repeat the same information for each species.

**Pathogenesis.** Most GI parasites produce a protein-losing gastroenteropathy. In severe cases, hypoalbuminemia may develop. Enteritis will induce changes in the secretory status of the intestine. Appetite and utilization of the feed consumed is reduced, depriving the body of vital nutrients. Absorption of calcium and phosphorus is depressed, causing, in turn, arrested skeletal development in the young animal. Selenium uptake is also retarded. Young animals are at greatest risk when affected by parasitism because no resistance has been developed to the invading organisms.

**Clinical signs.** There are peracute, acute, and chronic forms of most parasitic diseases. Death may be caused by overwhelming invasion of an organ or system, but usually parasitism results only in debilitation in varying degrees. Over a period of time, the body loses the ability to resist minor infectious agents, and a secondary infection may take the animal's life. Some degree of unthriftiness usually accompanies parasitism. The skin lacks vitality.

Emaciation may be seen in longstanding cases, a result of inappetence, leading to anorexia, combined with poor food utilization. Inappetence and poor food utilization also inhibit growth and maturation of parasitized young animals. Diarrhea is the most prominent sign of enteritis, but it is important to recognize that diarrhea need not always be present in parasitism, especially when larvae invade such tissues as the liver or lungs.

Anemia may be seen in heavy infestations, even with parasites that are not blood suckers. The cutting mouth parts used for attachment may result in leakage of plasma and cells from capillaries.

**Diagnosis.** The presence of one or more of the signs noted above should direct attention to a differential diagnosis, including parasitism. Unless adult parasites have already been seen in the feces, some type of fecal examination should be conducted to begin the process of diagnosis. Reference to standard texts will provide an explanation of the methodology for fecal examinations. Also see Chapter 13.

Tables 12.3 and 12.4 list sizes of male and female adult parasites, larvae, and eggs and location of the internal parasites in the body. Table 12.5 lists external parasites. Identification may be possible only to the genus level and perhaps only to the family. However, this will usually suffice to indicate methods of management and therapy.

A direct smear is used as a quick preliminary procedure to determine the presence of nematode eggs.

Various types of differential centrifugation or flotation are used to identify eggs, which are separated by the specific gravity unique to each parasite ova. Likewise, various counting procedures allow estimation of the parasite burden. Interpretation of these counts should be done by experienced persons.

Special methods, such as a Baerman apparatus, are required for detection of parasites that pass larvae in the feces instead of eggs (Dictyocaulus sp.). A final diagnostic tool is response to treatment. The clinician may choose to treat on the basis of suspicion and previous experience.

**Management.** The basic principles involved in the management and treatment of parasitism are identification of the parasite(s), at least to genus, and review of the life cycle. The same management procedures as have been described in the literature for similar parasitism in domestic livestock may be used in elephant gastrointestinal (GI) parasites.[92]

The nutritional status of the group should be evaluated and, if necessary, appropriate changes made.

Numerous anthelmintics are safe and effective against gastrointestinal nematodes in ruminants. See Table 12.6. The pharmacodynamics of most anthelmintics in elephants is unknown.[22,29] Until such information is available, the clinician should use equine doses and dosing intervals as a model. See Chapter 15 for more details on medication.

As in the case of domestic livestock, local populations of parasites in elephants may develop resistance to an anthelmintic or a class of anthelmintics.

When an animal is suffering from severe dermatitis associated with external parasitism, chemicals are more likely to be absorbed through the damaged skin, which

**Table 12.4.** Biological Data for Selected African Elephant Parasites

| Scientific Name | Common Name | Adult ♀ mm | Adult ♂ mm | Egg µ | Intermediate Host | Location in Elephant: Adult | Location in Elephant: Immature | Comments |
|---|---|---|---|---|---|---|---|---|
| **Nematodes** | | | | | | | | |
| *Murshidia linstowi* | GI nematode | 25.8–29.5 | 21.0–26.5 | 50 × 38 | None | Small intestines | | |
| *Murshidia africana* | Same | 18.0 | 17.0 | 62 × 32 | None | Stomach | | |
| *Murshidia africana* | Same | 17.0 | 17.0 | 60 × 35 | None | Large intestine | | |
| *Quilonia africana* | Same | 21.0 | 16.0 | 73 × 30 | None | Stomach | | |
| *Quilonia loxodontae* | Same | 26.0 | 24.0 | 75 × 38 | None | Intestines | | |
| *Amira sameera* | Same | 11.5–12.0 | 10.5 | 35 × 39 | None | Stomach | | |
| *Bunostomum brevispiculum* | Hookworm | ? | 12.1 | ? | None | | | |
| *Grammocephalus slathratus* | Bile duct hookworm | 36.0 | 45.5 | 50 × 35 | None | Bile ducts | | |
| *Leiperenia leiperi* | | 3.9 | 3.8 | 56 × 60 | None | Intestines | | |
| *Parabronema africanum* | Stomach worm | 57.0 | 40.0 | Viviparous | None | Stomach | | Causes ulcers, granulomas |
| *Loxodonofilaria* sp. | Microfilaria | | | | | Fascia of aortic arch and aorta | Blood | |
| **Trematodes** | | | | | | | | |
| *Brumptia bicaukdata* | | 7.0–9.0 wide 12.0–15.0 long | Bisexual | 114 × 76 | | Large intestine | | Eggs operculated |
| *Gastrodiscus aegypticus* | | 5.0–8.0 wide 7.0–8.0 long | Bisexual | 140 × 95 | | | | Eggs operculated |
| *Fasciola hepatica* | Liver fluke | 13.0 wide 30.0 long | Bisexual | 140 × 80 | Amphibious snails —*Lymnaea* spp. | Bile ducts | | |
| *Fasciola jacksoni* | Liver fluke | 9.0–12.5 wide 12.0–14.0 long | Bisexual | 115 × 58 | Amphibious snails | Bile ducts | Duodenum, abdominal cavity, penetrate liver | Eggs operculated |
| **Cestodes** | | | | | | | | |
| *Anaplocephala manubriata* | Tapeworm | 1.6 cm wide 2–6 cm long | Bisexual | 70 × 80 | Orobatid mites | Intestine | Intestine | |

**Table 12.5.** Biological Data of Selected Arthropod Parasites of Elephants

| | | Imago (Adult) | | Egg Size | 3rd Instar | |
|---|---|---|---|---|---|---|
| Scientific Name | Common Name | Length mm | Color | Length mm | Length mm | Comments |
| **Flies** | | | | | | |
| *Platycobboldia loxodontis* | Blue elephant stomach bot fly | 10.0–13.0 | Head is bright orange, body is metallic dark blue | | 10.0–23.0 | 3rd instar in stomach, puparium is brownish black 15–19 mm long |
| *Rodhainomia roverei* | Green elephant stomach bot fly | 11.0–14.0 | Green | 1.0 | 15.0+ | 3rd instar in stomach, puparium is 13–15 mm long |
| *Ruttenia loxodontis* | African elephant skin maggot | 7.0–13.0 | Body covered with long yellow hair | 0.5–0.25 | 7.0–10.0 | 3rd instar in boils in upper hind legs and flanks, puparium 9 mm long |
| *Neocuterebra squamosa* | African elephant foot fly | 14.0–17.0 | Metallic blue and violet | | 23.0 | 3rd instar in wounds |
| *Pharygobolus africanus* | African elephant throat bot fly | 13.0–15.0 | Black | | Up to 30.0 | 3rd instar in pharynx and upper esophagus |
| *Cobboldia elephantis* | Asian elephant stomach bot fly | 15.0– 20.0 | Head bright orange, body and thorax shiny black | 1.8–1.9 | 25.0+ | 3rd instar in stomach |
| *Elephantoloemus indicus* | Asian elephant skin maggot | 4.5– 6.0 | Body yellow to orange, black marks on thorax and abdomen | 1.0 | 9.0–18.0 | 3rd instar in wounds |
| *Chrysomya bezziana* | Old World screwworm, Bezzi''s blow fly | 8.0–12.0 | Metallic green or blue | 1.25 | 9.0–18.0 | Instars in viable tissue, flies not attracted to decomposing flesh |
| **Lice** | | | | | | |
| *Haematomyzus elephantis* | Elephant louse | 3.0 | Gray | 0.8 × 0.3 | | On skin |
| **Ticks** | | | | | | |
| *Amblyoma tholloni* | Elephant tick | | | | | |

may lead to the development of toxicosis. It is suggested that such animals be treated in stages.

A final management recommendation is for fecal samples to be monitored semiannually to evaluate the effectiveness of the management plan or medication. The examination should coincide with known exacerbation of parasite loads for the area.

## Order Rhabditida, Family Strongyloididae

**Identification.** This group of nematodes should not be confused with strongyles in the family Strongylidae.[13] *Strongyloides elephantis* was recovered from an Asian elephant imported into the United States.[49] Adult females measured 2.6 to 3.6 mm in length. The eggs measured 23 X 49µ.

**Life cycle.** The life cycle of *Strongyloides elephantis* is unknown. However, the life cycles of other *Strongyloides* spp. are unique among parasites of domestic animals by having alternate free-living and parasitic generations.[13]

A description from one of the ungulate species, *Strongyloides westeri*, follows: Parasitic males do not exist, nor does the parasitic female contain male gonads. She produces eggs by mitotic parthenogenesis and the larvae from these eggs are designated homogonic rhabditiform larvae to distinguish them from the heterogonic offspring of the free-living sexual generation.

The homogonic larvae may molt twice and become infective to an animal or molt four times to become free-living males and females. The free-living adults copulate and produce heterogonic rhabditiform larvae that molt and become infective filariform larvae.

The infective larvae may be ingested, but more commonly they penetrate the skin and undergo more molts, finally becoming a female, living primarily in mucosal crypts in the small intestine of the horse, pig or zebra and probably also the elephant.[13,101] The larvae may migrate to various tissues including the mammary gland and may be found in colostrum and milk: an important means of transmission.[13]

**Clinical signs.** The female parasites recovered from the elephant in the United States were part of an overwhelming intestinal parasite load, so the direct effects of the strongyloides could not be determined. In most adult animals there are no signs of strongyloidosis. However, in neonates and nurslings, massive infection may be lethal following acute or peracute enteritis and diarrhea.

**Table 12.6.** Anthelmintics and Other Parasiticides Used in Elephant Management

| Generic Name | Trade (Commercial) Names: North America | UK/Europe | Africa | India/SE Asia | Dose/Route mg/kg | Used Against |
|---|---|---|---|---|---|---|
| **Macrocyclic Lactones** | | | | | | |
| Ivermectin | Ivomec,[46] Eqvalan[46] | Eqvalan[46] | Ivomec,[45] Eqvalen | | Horse— 0.2—PO<br>Elephant—0.1—P.O.* | Ascarids, strongyles, grubs, lice, mites |
| Doramectin | Dectomax[53] | Dectomax[54] | | Dectomax[56] | Cattle—0.2—PO<br>Elephant—no dose | Strongyles, Sarcoptes |
| Moxidectin | Cyndectin,[19] Quest[19] | Quest[23] | | | Horse—0.3—PO<br>Elephant—no dose | Strongyles, stomach bots |
| **Benzimidazoles** | | | | | | |
| Albendazole | Valbazen[53] | Valbazen[48] | Valbazen[61] | Banmith[48] | Cattle—10.0—PO<br>Elephant—2.5—PO | Cestodes, trematodes, strongyles |
| Fenbendazole | Panacur[27] | Rintal[10] | Panacur[29] | Fenbezol,[59] Fenmor[26] | Horse— 6.0<br>Elephant—no dose | Strongyles, stomach bots |
| Fenbantel | Rintel[9] | Rintal[10] | Rintal[11] | | | |
| Thiabendazole | Equizole,[47] omnizole,[47] TBZ[47] | Equizole[13] | Equizole | | Horse—44.0—PO<br>Elephant—20.0— PO | Strongyles |
| Mebendazole | Telmin[27] | Telmin[65] | Vermox[30] | Mebendal[73] | Elephant—2.5 to 4.0—PO<br>6.0 to 7.0 PO for trematode | Strongyles, trematodes |
| Oxfenbendazole | Benzelmin | Benzelmin[65] | | | Horse—10.0—PO<br>Elephant—2.5—PO | Ascarids, strongyles |
| Oxibendazole | Anthecide[53] | Anthecide[54] | | | Horse—10.0—PO<br>Elephant—no dose | Ascarids, strongyles |
| **Imidazothiazole** | | | | | | |
| Levamisole HCl | Tramisol,[43] Ripercol L, Generic[65] | Generic[65] | Tramisal,[29] Levisol, Ripercol[35] | Almizol,[4] levamisole[74] | Cattle—5.5 to 10.0—PO<br>Elephant—2.5 to 3.0—PO | Strongyles, hookworms |
| **Tetrahydropyrimidines** | | | | | | |
| Pyrantel pamoate | Strongid T[53] | Strongid T[54] | Nemex,[55] combantrin[55] | | Horse—6.6 to 13.2—PO<br>Elephant—no dose | Ascarids, strongyles, cestodes |
| Pyrantel tartrate | Strongid C,[53] Baminth-48 | Banmith[54] | | | | |
| Morantel tartrate | Rumatel | N.A. | | | Cattle—9.68—PO<br>Elephant—2.0 to 4.0—PO | Strongyles |
| **Piperazines** | | | | | | |
| | | | | | Horse—14.4—PO<br>Elephant—no dose | Strongyles |
| Piperazine adipate | Generic[13] | Generic[13] | Wormol,[49] Piperazine[49] | Piperazine[59] | Horse—110.0 PO<br>Elephant—no dose | Ascarids |
| **Isoquinolenes** | | | | | | |
| Praziquantel | Droncit[9] | Droncit,[10] paratak | | Droncit,[12] Paratak[64] | Sheep—10.0 to 15.0—PO<br>Elephant—2.5 to 4.0—PO | Cestodes, trematodes |
| **Organophosphates/Carbamates** | | | | | | |
| Carbaryl | | | Karbadust | | Dusting powder | For external parasites |
| Fenthion | Tiguvon[9] | | Tiguvon-spoton[11] | | Cattle—3%—pour-on<br>Elephant—no dose | Flies, lice, grubs |
| Dichorovos | Atgard,[13] Task, Tridex paste | Atgard[13] | | | Swine—28.0 to 45.0—PO<br>Elephant—no dose | Ascarids, strongyles |
| **Miscellaneous** | | | | | | |
| Chloruson | Curatrem[47] | Curatrem[48] | Curatrem[17] | | Cattle—7.5—PO<br>Elephant—no dose | Trematodes |
| **Pyrethrins** | | | | | | |
| Permethrin | Permectrin2,[65] Atroban[65] | Generic | Generic | Pulvex, generic | Sprays, dips, shampoo, dust | Insecticide |

Superscript numbers in this table refer to the sources listed in Appendix 3.

N.A. = not available, PO = per orum (orally), SQ = subcutaneously, IM = intramuscularly. * = for an allometric dose based on weight of elephant.

**Diagnosis.** These parasites are tiny and may be overlooked in a casual necropsy examination. Finding eggs in the feces may be difficult, because they hatch to larvae quickly. Identification of the rhabdidiform and filariform larvae are the key to diagnosis.

**Management.** Strongyloid parasitism responds to most anthelmintics.

## Order Strongylidea

**Identification.** Most of the nematodes of elephants are classified in this order (see Tables 12.1 and 12.2). Generally these are unfamiliar to those having experience with domestic livestock. No species of Strongylus, Capillaria, Trichostrongylus, Trichinella, or Oxyurids are found in elephants. The genera of nematodes in the family Strongylidae affecting elephants include Choniangin,[79,80,114] Equinubria, and Decrusia.[83] The genera are the same for both Asian and African elephants, but generally the species differ (see Tables 12.1 and 12.2).

In the family Cyanthostomidae, the genera of the most common strongyles in elephants include Murshidia[10,19,65,66,82,83,114] and Quilonia[28,58,82] (see Tables 12.3 and 12.4).

**Life cycle.** Little is known of the life cycles of elephant strongyles. Presumably the cycles are similar to those of the strongyles of domestic livestock (Fig. 12.5).[13,69] Adults are found in the stomach, small intestine, cecum, and large intestine, depending on the species. Females produce fertilized eggs containing embryos in the morula stage. The eggs continue embryonating in the feces to L-1, still encased in the egg case. Under ideal conditions, hatching occurs in 1 to 2 days. The free-living L-1 larvae feed on microorganisms in the feces and molt to L-2 and again to L-3, which is the infective stage, taking 4 to 6 days.

L-3 larvae migrate out of the feces in about 1 week and climb onto vegetation. The life cycle is direct, and the elephant ingests plants containing infective L-3 larvae, which mature through L-4 and L-5 to become adults in the stomach or the intestine.

**Epizootiology.** Frequently the presence of gastrointestinal nematodes is simply reported as "strongyles" with no attempt made to identify further.[44] The length of time necessary for maturation of free-living larvae is dependent upon climate, season, temperature, and the species of strongyle. Some larvae will survive a mild win-

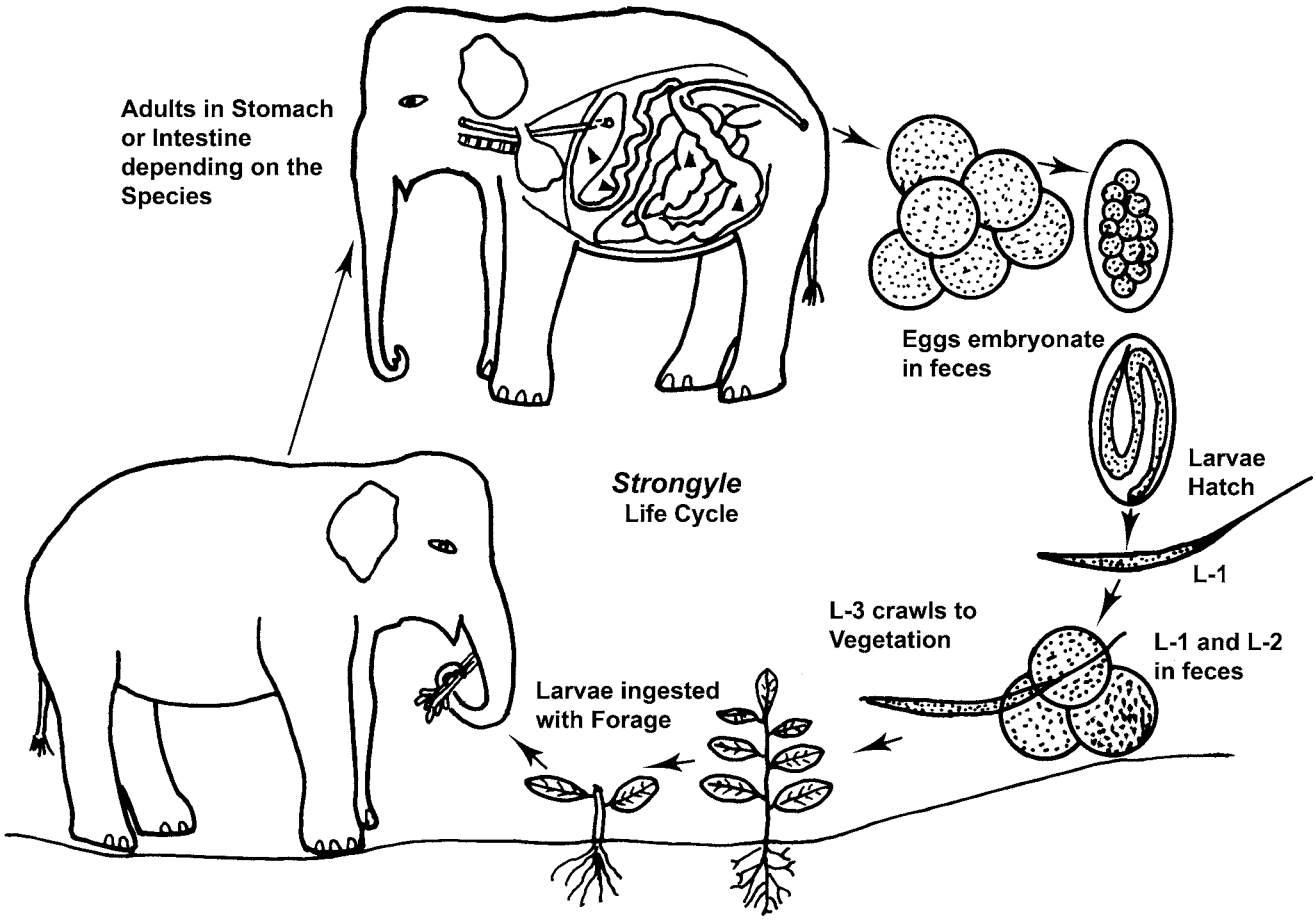


***Figure 12.5.*** *Typical life cycle of an elephant strongyle.*

ter in protected areas. Drought and desiccation are detrimental to the survival of most larvae, but some species have evolved in such environments and have developed adaptations for survival.

The clinician faced with a strongyle problem in a group of elephants should review one of the standard texts on the subject in horses and apply the same principles.

**Management.** Anthelmintics currently used in elephant strongylosis include fenbendazole, 5 mg/kg[60,89]; albendazole,[103] ivermectin, and mebendazole, see Chapter 15 for details of administration.

## Hookworms (Family Ancylostomidae)

**Identification.** Several species of hookworms in elephants include *Bathmostumum sangeri*, *Grammocephalus* spp., and *Bunostomum s*pp. See Chapter 31, Tables 12.1 and 12.2. *Bunostomum* spp are also found in sheep, but the other genera are unique to elephants.[77]

**Life cycle.** The life cycle of Bunostomum is direct. Adults are attached to the mucosa of the small intestine and are blood suckers. The eggs require a few days after passage in the feces before infective larvae are produced. These larvae may enter the body via the mouth or through the skin. If via the skin, the larvae migrate to the lung via the venous or lymphatic vessels and mature to L-3. These are coughed up and swallowed. L-4 larvae migrate to the intestine. The prepatent period is 30 to 56 days.

**Epizootiology.** Infective larvae are susceptible to desiccation, so this parasite is a problem only where there is permanent moisture or high humidity. There is no encystment in the muscles, nor is there transmammary migration, as occurs in ancylostomiasis.

**Diagnosis.** Bunostomum hookworms inhabit the intestine. Generally the eggs may be observed on fecal flotation. Lesions at necropsy vary from pale mucous membranes to punctate hemorrhages of the mucosa to loose watery ingesta in the intestine and the presence of numerous hookworms.

## Grammocephalus (Family Ancylostomidae)

**Identification.** These are unique hookworms in that they inhabit the bile ducts. *Grammocephalus clathrates* is found in the African elephant, including individuals imported into the United States,[3,31,37] and *Grammocephalus varedatus* in the Indian elephant; see Table 12.2.

**Life cycle.** The precise life cycle is unknown, but is presumably similar to *Bunostomum* spp. hookworms (Fig. 12.6).

Adults are found in medium to large or main bile ducts.

**Clinical signs.** Adult parasites are blood suckers, so anemia and weakness plus other signs of hepatic insufficiency occur in severe infestations.

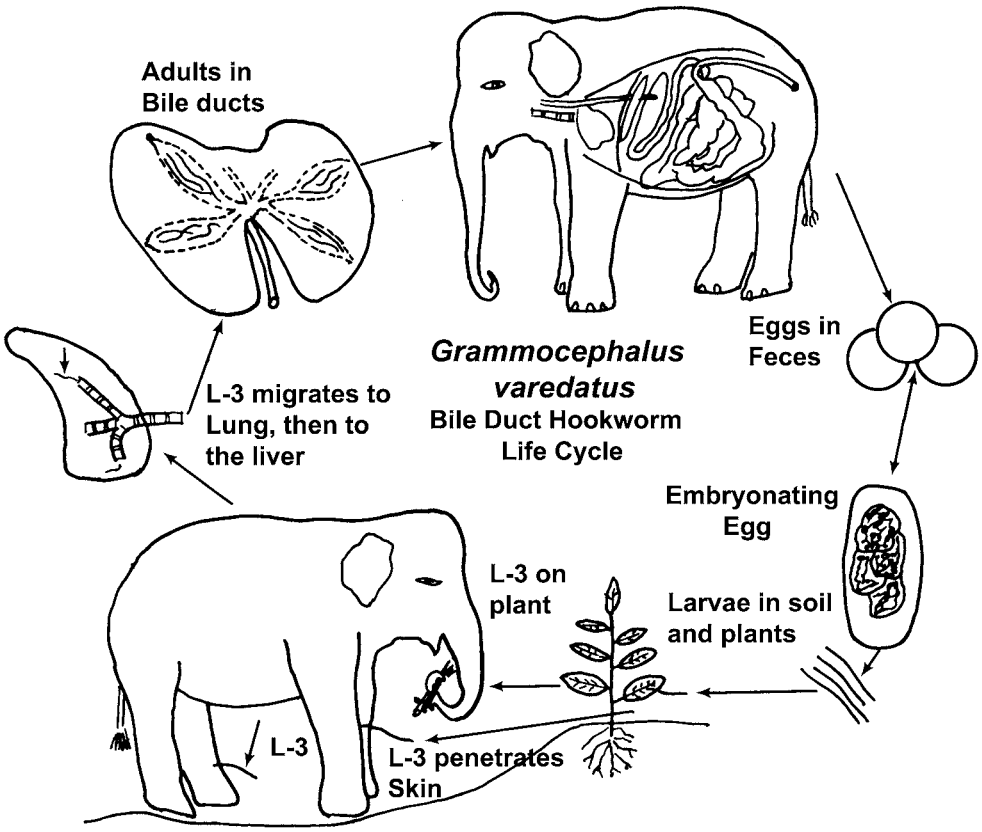


***Figure 12.6.*** *Life cycle of the bile duct hookworm* Grammocephalus varedatus.

**Diagnosis.** The eggs are similar to Bunostomum eggs, which are irregularly spherical with blunt ends. At necropsy, gross examination reveals petechia, erosions, ulcers, and necrotic foci in the bile ducts. In elephants the pancreatic duct anastomoses with the main bile duct 5.0–8.0 cm before emptying into the duodenum. Pancreatitis may result from severe hookworm infestation. Histologically there are epithelial hyperplasia and inflammatory infiltrates of eosinophils and round cells. Additionally ulcerations and microabscesses were seen.[6]

## Gapeworms (Family Syngamidae)

Elephants have one species, *Mammonogamus indicus,* found in the Asian elephant.

## Family Atractidae

Leiperenia is the only genus parasitizing mammals. *L. moreli* is found in the intestine of African elephants and *L. galebi* is found in Asian elephants. Virtually nothing is known of the life cycle or clinical signs.[20]

## Ascarids (Order Ascarididea, Family Ascaridae)

Asian elephants are known to harbor a unique ascarid, *Toxocara elephantis*. This is a rare parasite in elephants and little is known about the life cycle or clinical signs.

## Parabronema (Order Spiruridea, Family Acuaridae)

**Identification.** Two species are found in the Asian elephant, *Parabronema indicum* and *P. smithi,* and three in the African elephant, *P. africanum, P. rhodesiense,* and *P. longispiculatum*.[47]

**Life cycle.** The complete life cycle is unknown, but it is likely direct (Fig. 12.7).[101] The female is viviparous, so no eggs are found in the feces. Presumably L–3 larvae leave the feces and attach to vegetation, which is then ingested by an elephant.

**Clinical signs.** Signs are variable, but they are similar to those of other gastrointestinal nematodes. The nematodes invade the crypts in the stomach causing squamous metaplasia and pustules and resulting in necrotic suppurative ulcers, all of which interfere with digestion.

**Diagnosis.** Diagnosticians must use a Baerman apparatus to detect larvae in the feces. At necropsy, the stomach may be covered with ulcers varying in size from 0.3–5.0 cm. The margins of the ulcers are elevated by the production of granulation tissue.[6] Parasites that invade the crypts cause squamous metaplasia of the mucosa along with pustules and necrosis and ultimately the production of ulcers. The parasites may be observed on the surface of the ulcers and on nonaffected mucosa.

## Dipetalonema (Order Filaridea, Family Dipetalonematidae)

**Identification.** Two species of Dipetalonema parasitize African elephants: *Dipetalonema loxodontis* and *D. gossi*. *D. gossi* is a long (15 cm), slender filarial nematode.[5] In a study conducted in Uganda during a culling operation, positive observations of microfilaria in the blood were 31% of young elephants, but as high as 87% in adult animals.[69,115] Asian elephants may be parasitized with *Indofilaria parabiramani* and *Dipetalonema asciatica.*[4,95] Microfilarial dermatitis around the toenail bed and

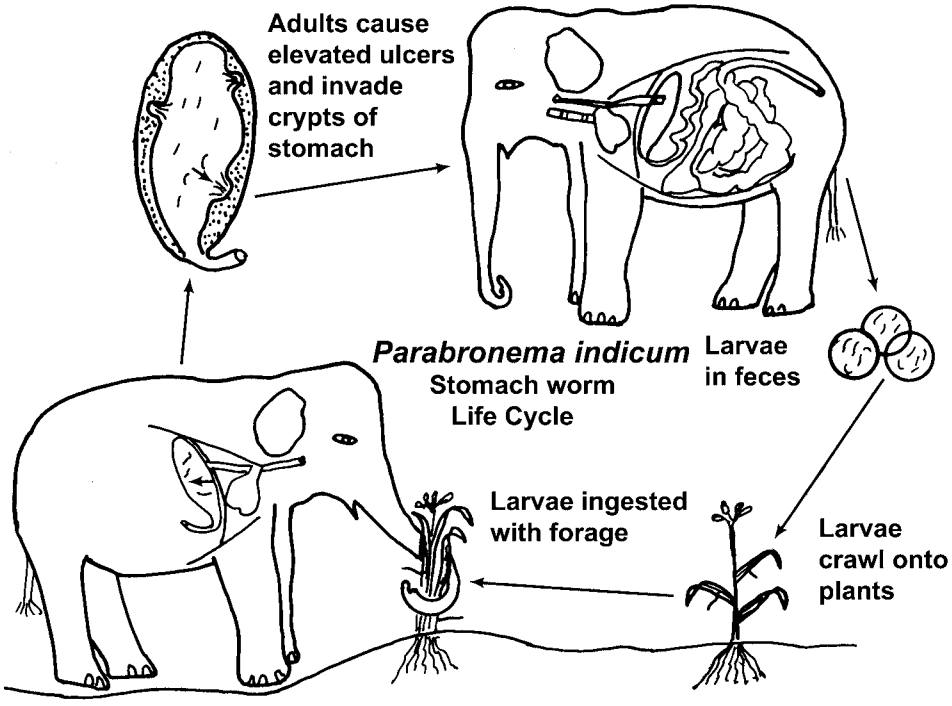


***Figure 12.7.*** *Life cycle of an elephant stomach worm* Parabronema indicum.

heels of Asian elephants has been ascribed to an unidentified *Stephanofilaria* sp.[1,11,69,107] Another report indicates that the Stephanofilaria is indistinguishable from *S. assamensis*.[27]

**Life cycle.** Little is known of the life cycle of filarids in elephants. If similar to filarids in other species, the adult female produces first stage larvae (microfilariae), which circulate in blood. Blood-sucking insects are the intermediate host where the larvae develop to third stage larvae. The infective larvae are deposited on the skin of the elephant when the insect takes a blood meal. The larvae, after entering the host through the bite site, continue development through the fourth and fifth stages in the blood.[13] The adults are found in various locations. In *Dipetalonema loxodontis,* adults are in the hepatic portal venous system.

**Clinical signs.** Signs are those of a hepatic insufficiency.

**Diagnosis.** Gross pathology in African elephants dying from this parasitism revealed pinpoint, yellowish-grey pustules on the edge of the liver. Vascular changes involved the portal venous system, hepatic veins, and centrolobular veins. Vessels were thickened and inflamed and contained early or organized thrombi, which may be calcified. Dead, intravascular filarids were observed in 50% of the elephants necropsied.

Microscopically there were fibrinous thrombosis and an eosinophilic inflammation with smooth muscle hypertrophy, hyperplasia, and fibrosis.[6]

**Hemorrhagic dermatitis.**

Identification. *Indofilaria pattabiramani* is a filarid parasite of Asian elephants in India and Myanmar. The male of this species has not been found. The female is 79 mm long by 0.765 mm in diameter at the thickest area, with tapering at both ends.[4]

Life cycle. The female is located in nodules of subcutaneous tissue on the sides and ventral aspect of the abdomen and less frequently on the neck, chest, and outer aspect of the thighs. As many as 270 nodules have been counted on a single elephant.

Each nodule is 1–2 cm in diameter and approximately 1 cm thick. A day or two after the appearance of the soft-whitish nodule, an opening is seen in the center of the nodule through which blood oozes. Up to 10 ml of blood may exude from each nodule. The nodules may persist for nearly 4 years, with older nodules being firmer (caused by fibrosis).

The female filarid is viviparous. Microfilaria within the uterine tubes are sheathed, but those in the peripheral circulation and from the blood oozing from the nodule are unsheathed. Microfilaria vary from 0.137–255 mm, and 0.007–0.011 mm in diameter. The microfilaria are concentrated in the blood oozing from the nodule.

Presumably blood-sucking insects transmit the filarial worm from one elephant to another. The cycle in the insect is unknown.[4]

Clinical signs. The nodules are evident, as are the openings from which the blood oozes. The disease is described as a "hemorrhagic dermatitis."

Diagnosis. Diagnosis is by identification of the microfilaria. A procedure for detecting microfilaria in nocturnally collected peripheral blood samples is described in the Myanmar section of Chapter 35.

Management. There are no reports on treating elephants infested with this parasite, but ivermectin should be effective.

### *Trichuris* sp. (Order Enoplida, Superfamily Trichinelloidea)

There is only a single report of this parasite in elephants. A 27-year-old Asian bull in a major zoo in Germany was found dead in the morning. At necropsy he had a marked hepatopathy, but also a massive infestation of whipworms.[94]

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# 13 Antemortem Diagnostics

## Section I: General Techniques

Susan K. Mikota

### INTRODUCTION

With some exceptions imposed by the elephant's large mass, most diagnostic techniques used in other large animals may be applied to elephants. This chapter discusses standard diagnostic techniques as well as those under investigation. This discussion is divided into two sections. SECTION I describes a variety of general techniques, presented in alphabetical order, with reference to their specific use in elephants where such information has been published. SECTION II is devoted to radiology.

### BIOPSY

Biopsy techniques will vary with the tissue to be sampled and are best modeled after procedures used for other large animals because little information specific to elephants has been published. A technique for obtaining a paralumbar kidney biopsy has been described using a modified 15-inch, 8-guage biopsy needle. The procedure was performed on an 8-year-old African elephant.[22] Bone marrow may be sampled from the dorsal spinous process of L-1 using a Jamshidi needle (Baxter Hospital Supply Division, Deerfield IL 60015, USA) inserted through a stab incision.[1] General anesthesia is required. A remote method for obtaining skin biopsy samples using a modified dart (Palmer Cap-Chur Inc., Powder Springs, Georgia 30127, www.palmer-capchur.com) has been developed and has potential application for collecting samples from free-ranging elephants.[22] Advances in laparoscopy (discussed below) will facilitate greater use of biopsy as a diagnostic technique.

### CULTURE

Proper sample collection is essential if meaningful diagnostic information is to be obtained. Samples should be collected aseptically and labeled clearly. Commercial culture swabs are adequate for most purposes. Swabs may be refrigerated overnight but should not be stored for longer periods.

When sampling from a wound, clean the surrounding area and remove any superficial debris. Sample abscesses by aspirating deeply with a syringe and large bore needle or after lancing (purulent material may be quite thick). Abscesses and deep wounds should be cultured for anaerobes. This requires special collection devices that maintain the sample in an anaerobic environment during transport. Liquids or pus may be held anaerobically in a syringe if the air is expelled and the needle is plugged. Consult your laboratory for specific instructions regarding sample collection and shipping, especially when anaerobic, fungal, mycoplasma, or viral infection is suspected.

Proper packing of infectious biological samples is essential. Use leakproof, sterile, unbreakable containers for liquids or tissues, especially if zoonotic diseases are suspected. Seal with para-film (Fisher Scientific, Allentown, Pennsylvania, 18106, www.fisherscientific.com) (or duct tape if necessary) and place in heavy zipper-lock plastic bags. Double or triple bagging is advised. A sponge may be placed in the shipping container to absorb fluids in the event a leak occurs. Include appropriate forms (place in plastic zipper-lock bags). Verify that your overnight carrier does not irradiate packages. Commercial shippers are available that are specifically

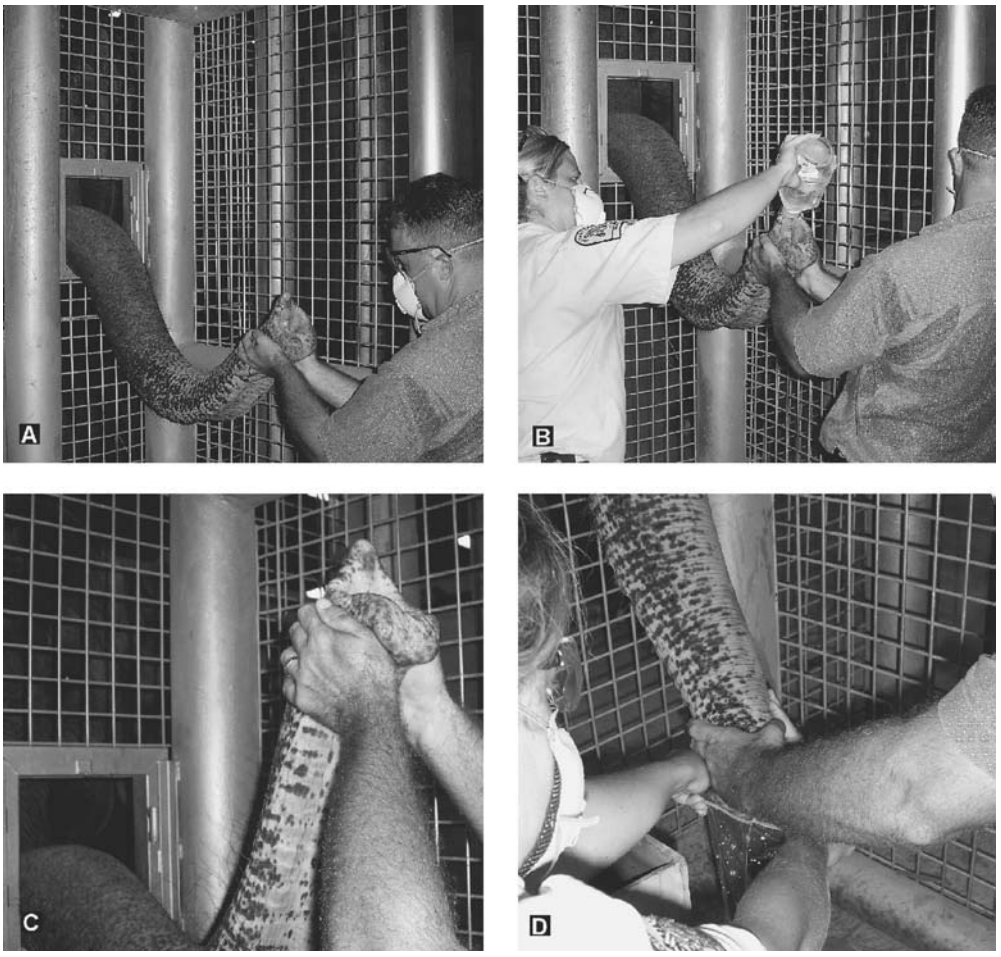


***Figure 13.1.*** *Procedure for obtaining a trunk wash sample for mycobacterial culture. A) Elephants must first be conditioned to accept manipulation of their trunk. B) Sterile saline is instilled. C) The trunk is raised to distribute the wash. D) The trunk is lowered and the sample is collected in a zipper lock plastic bag. Training elephants to exhale forcibly is desirable because this may facilitate collecting a sample from lower in the respiratory tract. Samples should be transferred to screw top plastic vials and packaged securely before submitting to the laboratory. (Photos courtesy of Busch Gardens, Tampa.)*

designed for biomedical samples and which are in compliance with current federal and international regulations (Nomadic® shipper; www.thermosafe.com).

Although organisms associated with pathology are frequently reported, there has been little attempt to determine normal microflora in healthy elephants. An exception is a study of conjunctival microflora in clinically normal elephants. Gram-positive bacteria (mainly *Staphylococcus* and *Corynebacterium* spp.) comprised the most frequent isolates.[42]

### Trunk Wash

A special procedure has been developed to collect samples for mycobacterial culture[20] (Fig. 13.1). Sterile saline (~60cc) is flushed into one nostril, the trunk is elevated, and then it is lowered into a 1-gallon zipper-lock plastic bag or other collection device. Elephants must be trained to accept this procedure, and many may also be taught to exhale forcibly, which enhances sample collection from lower in the respiratory tract. Although mycobacteria have been isolated from swabs and nasal exudates, collection of samples by the trunk wash technique is preferred. Due to intermittent shedding, a series of three samples are required under the 2003 U.S. Guidelines.[44] Tuberculosis in elephants is discussed in Chapter 11.

## CYTOLOGY

The microscopic evaluation of cells may be a useful aid to diagnosis. Examination may be in-house if equipment and expertise are available, or samples may be submitted to commercial laboratories. Samples may be collected by swabbing or scraping if the area of interest is solid (e.g., skin lesion) or by aspiration if fluid (e.g., cyst, abscess). Cytology may be used to evaluate urine, vaginal or other discharges, and joint or other body fluids. Samples should be collected using proper aseptic technique. Specific procedures for sample collection, processing, and staining will vary with the tissue under investigation, the diagnostic goal, and recommendations from the consulting laboratory. When submitting slides it is advisable to leave some unstained and to retain a duplicate set. Details of sample collection and processing are beyond the discussion here and appropriate references should be consulted. Proper preparation for shipment to prevent breakage or leakage should not be overlooked.

Although cytology is undoubtedly used in elephants, reports have been limited to largely unsuccessful attempts to use vaginal cytology as an indicator of the estrous cycle.[5,19,21,39]

## ELECTROCARDIOGRAMS (ECGS)

ECGs are discussed and illustrated in Chapter 24.

## FECAL EXAMINATION

### Parasites

Microscopic examination of feces to detect eggs, cysts, or larvae is typically used to diagnose intestinal parasitism. Knowledge of parasites common to the geographic area is essential in selecting the appropriate method, based on the ease of recovery with flotation solutions of different specific gravities (SG). Although the eggs of most intestinal parasites can be detected using flotation techniques, sedimentation procedures may be required when flukes are suspected because many fluke eggs are denser than common flotation media, and thus are not easily recovered by these methods. Some fluke eggs do float, but several do not.

Fecal examination may be performed qualitatively to determine the presence of parasites in an individual or quantitatively to determine the level of parasitism in a herd. Always use as fresh a sample as possible. The high proportion of undigested fibrous material in elephant feces makes it more difficult to obtain an adequate sample, and the voluminous amount of feces that are passed may dilute the concentration of parasites. These factors should be considered when selecting the techniques to be used. Samples may be concentrated by using screens or mesh to hold back much of the fibrous debris as the water and parasitic structure pass into a collection vessel. This is allowed to stand for 2 hours and decanted carefully until a small amount remains. The remaining quantity can then be used in flotations.

**Flotation and sedimentation techniques.** The direct smear, in which a small amount of feces is added to a drop of saline on a microscope slide, is not practical for elephants. Although it may be possible to detect parasite eggs, the sample size is too small to engender confidence that parasites are absent if no eggs are observed.

A simple flotation is performed by mixing feces in a small vial with a flotation medium. Recipes for several flotation solutions are listed in Table 13.1. Sugar solutions are undesirable for passive floatation because the viscosity often impedes the recovery of many eggs; however, they are excellent for centrifugation techniques (personal communication, Cliff Monahan, DVM, PhD, Ohio State University, April 2005). Also, unlike salt solutions, they don't crystallize, so slides can be stored for many days (under refrigeration) if further examination is needed. A coverslip is placed on top, and after 15 minutes the coverslip is transferred to a slide and examined. This is the standard technique used for most domestic species. A variation (the Willis technique) that uses a larger amount of feces and eliminates bulky debris is recommended for elephants. Fill a paper cup or glass jar one-third to one-half full with feces, add flotation solution and mix thoroughly. Strain through a tea strainer into the flotation vial and proceed as above.

**Table 13.1.** Composition of Flotation Solutions[a]

Sodium chloride: 311 g NaCl dissolved in 1 liter of water; specific gravity (SG)[b] 1.20.
Sugar solution[c]: 1170 g table sugar in 1 liter of water; SG 1.2.
Sodium nitrate: 338–616 g $NaNO_3$ in 1 liter water; SG 1.2–1.33.
*Note that a higher SG exerts greater osmotic effect and may shrink cells; lower SG may be preferable for passive flotation.*
Zinc sulfate: 492.5 g $ZnSO_4 7H_2O$ in 1 liter water; SG 1.18–1.2.
May be preferable to $NaNO_3$ but more expensive.
Phosphate Buffered Saline (PBS): 8.0 g NaCl, 0.2 g KCl, 1.44 g $Na_2HPO_4$ in 800 ml distilled water qs to one liter, pH 7.2

[a]Personal communication, Cliff Monahan, DVM, PhD, Ohio State University, Columbus, Ohio, USA, April 2005.
[b]Verification of specific gravity is useful because some salts may absorb water vapor from the air and increase their weight.
[c] It is advisable to add a preservative such as 0.5% formalin (~9ml) to sugar-based solutions to prevent bacterial growth.

Centrifugation may also be used to concentrate the sample. Place the feces in a cup or jar and mix with water to wash. Strain as above to remove debris, pour into a centrifuge tube and centrifuge for 3–5 minutes at ~1000 rpm. Discard the supernatant, mix the pellet thoroughly in flotation solution filling the tube almost to the top, and centrifuge again for 5 minutes. Transfer the tube to a rack, add enough solution to create a slightly bulging meniscus, and place a coverslip on top. Wait 10 minutes then examine. Do not create a large meniscus that spills over the edge when placing the coverslip on top, because this fraction of the tube contains all the structures of interest. This technique is especially useful in cases of watery diarrhea (straining may not be necessary but still perform the wash step).

Trematode eggs are heavy and many will not float with conventional solutions, so if flukes are suspected, it is essential to perform a sedimentation technique. The formalin-ethyl acetate technique will recover trematode eggs as well as cysts, eggs, larvae, and any other parasitic elements present.

The procedure is outlined in Table 13.2. Commercial kits containing reusable polypropylene centrifuge tubes, screens, and caps are available and can be used for this ("ParaPak," Meridean Bioscience, Cincinnati, Ohio, USA, 1-800-543-1980, www.meridianbioscience.com).

The following is a simple method that may be used for the diagnosis of such heavy trematode eggs, including *Fasciola hepatica* (personal communication, Cliff Monahan, DVM, PhD, Ohio State University, April 2005).

Mix the fecal sample in a jar using water. Pour this mixture through a strainer or screen to hold back fibrous material. Strain into a ~250 ml pilsner glass (a funnel-shaped beer glass). A beaker or other container may

**Table 13.2.** Formalin-Ethyl Acetate Sedimentation Procedure[a]

1. Place the fecal sample in a jar with a lid. Mix with PBS, add 5% or 10% formalin in a ratio of 3:1 formalin:feces (this will kill any human pathogens, such as salmonella, that may be present). Wait 20 minutes.
2. Strain the sample into a polypropylene tube[b], adding more PBS if needed to bring the volume to 10 ml.
3. Add 3 ml ethyl acetate, cap, and shake vigorously.
4. Centrifuge for 5–10 minutes at 1000 rpm.
5. Remove the cap and free the plug from the centrifuge tube wall with a stir stick.
6. Decant the supernatant and ethyl acetate plug into a glass container for disposal.
7. Add water (or PBS if you are looking for trophozoites) to wash the pellet and centrifuge 3–5 minutes.
8. Decant the supernatant, add a small amount of saline, and resuspend the pellet.
9. Place a small amount of the suspension on a slide, apply a cover slip and examine.

[a]Personal communication Cliff Monahan, DVM, PhD, Ohio State University, Columbus, Ohio, USA, April 2005.

[b]It is essential to use polypropylene tubes; other tubes may melt upon contact with ethyl acetate.

be used; however, a container with a narrow bottom will help concentrate the sediment. Add water or saline and mix thoroughly. Wait ~2 minutes for heavy debris to settle and then decant the supernatant into a second glass. Allow at least 2 hours for the sample to sediment and then carefully decant the supernatant. Pour the sediment into a petri dish and examine with a dissecting microscope.

**Quantitative techniques.** Quantitative techniques are used as a measure of relative parasite burden and to determine pre- and posttreatment anthelmintic efficacy in herd situations. They have little application to individual animals because the eggs per gram (EPG) of feces are affected by numerous variables and do not correlate directly with the number of adult parasites. Quantitative techniques may have application to larger groups of elephants maintained in grazing areas where recurrent infection with strongyles is a problem. Serial evaluations may be used to determine appropriate deworming intervals to keep the parasite load low and reduce pasture contamination.

The two commonly used quantitative techniques are the Stoll's and the McMaster's. The latter is more accurate when the EPG is high (>1500) and the former when the EPG is low (<1000) (personal communication, Cliff Monahan, DVM, PhD, Ohio State University, April 2005). The Stoll's technique requires a centrifuge and the McMaster's requires a specialized counting chamber (Chalex Corporation, Issaquah, WA 98029 USA, 425-391-1169, www.vetslides.com). In these techniques the feces are weighed and diluted with a known quantity of solution. In the McMasters procedure, 4 grams of feces are diluted in 56 ml of liquid to yield a total volume of 60 ml and the eggs in 0.15 ml are counted. This count is equivalent to the number of eggs in one–one-hundredth of a gram of feces and is multiplied by 10 to yield the EPG.

It is quite likely that 4 grams may not be a representative sample from an elephant, given that a typical Asian elephant fecal bolus weighs 1.0–2.5 kg[2] and contains large amounts of fibrous material. A larger amount of feces and a greater volume of flotation solution may be appropriate when applying these techniques to elephants as long as the 1:15 dilution factor is maintained. Larger fecal sample volumes provide a more reliable outcome because the distribution of eggs in feces is not uniform; thus the larger amount used, the greater the confidence that this represents an accurate count.

Alternatively, samples may be submitted to a diagnostic laboratory. Commercial veterinary diagnostic laboratories may be available in large cities and may be found in veterinary schools in most countries. Submit one-fourth to one-half of a fresh fecal ball. Turn a plastic bag inside out over a hand and grasp the feces to be submitted. Then evert the plastic bag back over the hand and seal the bag. If done correctly, the outside of the plastic bag should not be contaminated. Place the filled bag in another sealable plastic bag containing information regarding the name, age, and sex of the elephant and contact information of the owner or submitter. Any pertinent clinical information is likewise helpful. Some states in the United States require that biological samples must be triple-bagged.

If samples must be shipped, the feces should be mixed into a transport medium such as SAF solution (sodium acetate, acetic acid and formalin). A human transport kit contains the solution in a screw-top sealable vial (Para-Fix, Medical Chemical Corporation, 19430 Van Ness Ave., Torrance, California, USA 90501, T: 1-800-424-9394, FAX: 1-310-787-4464). Each vial contains 15 ml of the solution. A small scoop is attached to the cap. Feces are added to the vial until the fluid level reaches a red line. This gives a 3:1 ratio of the fixative to the feces. The scoop is used to stir the feces into the solution. Then the container is capped and shaken until a homogeneous slurry is produced. Because the quantity of feces would be tiny for an elephant, several vials should be submitted from the same elephant.

Gross parasite specimens that have been passed in the feces may be fixed in 70% ethyl alcohol or 10% formalin. Helminths become more brittle in formalin. If specimens are small enough they may be placed in a red-topped serum tube. The stopper must be punctured with a needle to allow air to leave the vial when the stopper is pressed into the vial. The stopper should be capped with a paraffin sheet (or duct tape). Then the vial is placed in a sealable plastic bag and placed in a rigid mailing tube along with appropriate information.

### Fecal Steroid

Cortisol, testosterone, progesterone, estrogen, and other steroids may be detected in the feces. Analyses of fecal steroids have been used to study the female reproductive cycle, [9,16,45] male reproductive function, musth,[11,12] and stress.[46] These topics are discussed in detail in other chapters.

## HEMATOLOGY

Blood collection and cell identification are discussed in Chapter 25. Commercial laboratories are commonly used. In situations where manual techniques must be used, appropriate laboratory and clinical pathology texts should be consulted.

## LAPAROSCOPY

Laparascopy is a commonly used diagnostic technique in domestic large animals. Anatomical constraints, surgical complications, anesthetic considerations, and the need for specially designed instrumentation have limited its application in large megavertebrates such as the elephant.[38] Laparoscopy is a minimally invasive technique that may be used to perform organ biopsies and also has potential surgical application for procedures such as castrations or tubal ligations. A multiinstitutional collaborative effort has been initiated to address the design of laparoscopic telescopes of suitable length and durability, methods for abdominal insufflation, and the development of appropriate surgical approaches and anesthetic protocols for elephants and rhinoceros.[50] Laparascopic equipment for megavertebrates is not commercially available at this time. Veterinarians at the San Diego Wild Animal Park or Disney's Animal Kingdom may be contacted for further information.

## RADIOLOGY

See "SECTION II: RADIATION."

## SCHIRMER TEAR TEST

The Schirmer tear test may be performed without topical anesthesia using 5 × 80 mm test strips made from No.42 Whatman filter paper, notched at 5 mm from one end. The folded, notched end is placed in the lower conjunctival sac for 1 minute with the eyelids held closed. The wet length is measured in millimeters. Values for clinically normal Asian elephants range from 14–70 mm/min (mean 34.3 ± 1.7 mm/min), with the lowest values observed in elephants ≤20 years of age and highest values in elephants 41–60 years old.[43] Decreased tear production may be associated with keratoconjunctivitis sicca and increased production with injury or pain.

## SERUM BIOCHEMISTRY ANALYSIS

Serum biochemistry analysis is discussed in Chapter 25.

## SERODIAGNOSTIC TESTS

### Validity of Serodiagnostic in Elephants

The management and regulation of infectious diseases in livestock, horses, and companion animals is dependent upon the use of data from serologic and other laboratory diagnostic procedures. Many of these same tests are being used in wild animals, including elephants, to diagnose disease and to determine antemortem exposure to infectious agents. Utilization of diagnostic tests developed for one species in a different species for which the tests have not been validated, may be unwise. The assumption that a serological test will perform identically in elephants as it does in livestock may be incorrect.[13,17] There may be differences in pathogenic strains, serovars, host responses, and exposure to organisms of similar antigenic structure that produce cross-reacting antibodies. Furthermore, some assays require species-specific reagents/test components that may not be commercially available for elephants, and most assays have not been standardized.[13] Many of the diagnostic tests used in livestock have, by tradition, been accepted as valid in elephants; but in reality none has been adequately validated.[13,17]

*Accuracy (validity)* is the ability of a diagnostic test to produce the correct results. Sensitivity and specificity are measures of accuracy. *Sensitivity* is the probability that a test will correctly identify infected animals (true positives). A positive result using a test with a high sensitivity is a good indication that the animal is infected. On the other hand, false negatives may occur when 1) a recently infected animal has a low concentration of antibodies, 2) an animal is immunologically tolerant to infection, 3) the cutoff for the test was set too high or 4) the test was not correctly performed.

*Specificity* is the probability that a test will correctly identify animals that do not have the disease under evaluation (true negatives). False-positive reactions may occur under any of the following conditions: 1) the presence of specific and nonspecific antibodies following vaccination, 2) errors in handling and testing in the laboratory, 3) excessively low assay cutoff values, 4) cross-reactions with antibodies produced by antigenically related organisms.[13]

There is a vital need to validate tests used to evaluate disease in elephants. Validation of diagnostic tests is complex, and a detailed discussion is beyond the scope of this chapter. It should be understood, however, that antibody response to an antigen does not necessarily equate to disease or a carrier state in that animal. The accuracy of a diagnostic test is often measured by comparison to a "gold standard" (or reference test). Gold standards are not always perfect or may not exist. If the

sensitivity and specificity of the reference standard is poor or unknown, statistical analyses may be used to evaluate new tests.[8] Test validation may be further influenced by epidemiologic factors that vary within and among animal populations.[15] Guidelines for veterinary assay validation have been published and are continually being updated.[35,36]

Selected serodiagnostic assays are presented in Table 13.3. The test procedure is briefly described and examples of diseases that the method detects are listed.

## TEMPERATURE, PULSE, AND RESPIRATION (TPR)

Temperature, pulse, and respiration should not be overlooked as diagnostic techniques. Normal parameters are temperature 36–37°C (97–99°F); pulse 25–30 beats/minute, standing (72–98 in lateral recumbency); and respiration 4–12/minute. Sequential monitoring of these vital signs is recommended in ill elephants.

## THERMAL IMAGING (THERMOGRAPHY)

Thermography is a noninvasive technique that detects and measures the surface temperature (infrared radiation) of animate or inanimate objects. It has numerous industrial uses. Its application to human and veterinary medicine is based on correlating skin temperature variations with underlying pathological processes. It can be an adjunct to other diagnostic methods and a means to monitor the progression and response to therapy of identified lesions.

Surface skin temperature is affected by the contour of the body, underlying tissues and vascularity, metabolic rate, and other factors. Thermography has been used in humans to localize biopsy sites, assess blood flow and healing of skin grafts, and identify tumors. In veterinary medicine, thermography is used most frequently in horses to detect inflammation; identify musculoskeletal lesions, such as hairline fractures, tendon and ligament injuries, laminitis, joint disease, and muscle atrophy; and infections.[14] Lesions appear as "hot spots" or asymmetrical temperature differentials. Temperature differences greater than 1–2°C (1.8–3.6°F) may be significant.

Thermal imagers have advanced from large units connected to a liquid nitrogen source to highly portable handheld units. Contacting thermographic scanners must be placed on the surface being evaluated, but noncontact scanners detect invisible infrared radiation from a distance. The latter are typically thermographic cameras that convert the emitted infrared radiation to an electronic signal that is then transformed into a visual image. Thermal images can be displayed as stills or videos and can be downloaded and stored in a computer. The thermogram is essentially a photographic map of variations in surface skin temperature. A thermal image of an elephant is depicted in Figure 13.2 (see Color Section).

Thermography has been instrumental in furthering

**Table 13.3.** Selected Serodiagnostic Tests (Information is derived from a variety of sources for use as a general guideline. Consult other sources for specific test details.)

| Test | What It Detects | Basic Technique | Comments/Examples of Diseases Detected* |
|---|---|---|---|
| Complement Fixation Test (CF) | antibody (Ab) | A known amount of Ag is mixed with serial dilutions of test serum; complement is added. if Ab is present, an Ag-Ab complex forms and binds complement. Sheep RBCs coated with anti-sheep RBC Ab are added. If test serum contains Abs the sheep RBCs will not be lysed; if Abs are absent complement is free and will lyse the RBCs. | Primarily detects IgM. The ELISA is a simpler alternative. Examples: foot and mouth disease, mycoplasmosis. |
| Enzyme-Linked Immunosorbent Assay (ELISA) | antigen (Ag) or antibody | Direct ELISA: Ag is adsorbed onto a solid polystyrene surface. Serial dilutions of the test serum are added and then washed. Specific enzyme-linked Ab is added which binds to the Ag-Ab complexes creating a sandwich effect. Substrate for the enzyme is added producing a detectable color change that can be measured. Indirect ELISA: Ab is adsorbed to the plate. An enzyme-linked anti-Ab is added followed by the enzyme substrate. | Variations include competitive and kinetic ELISA. The ELISA is used for a wide variety of bacterial, viral, and parasitic diseases. Examples: foot and mouth disease, bluetongue, rinderpest, leptospirosis, anthrax, TB (experimental). |
| Fluorescent Antibody (FA) | antigen | Direct (DFA): Fluorescein-tagged Ab is applied to the test sample (e.g., tissue or blood) and then washed. Ag-Ab complexes, if present, will appear bright green under a UV microscope. Indirect (IFA): Ab is applied to the test sample and then washed; fluorescein-tagged specific antiglobulin is added. The tagged antiglobulin will attach to the viral-bound unlabeled Ab if complexes have formed and will fluoresce, confirming the presence of Ag. | DFA is quicker; IFA is more sensitive and specific; useful test for viral diagnosis. Example: rabies. |

**Table 13.3.** Selected Serodiagnostic Tests (Information is derived from a variety of sources for use as a general guideline. Consult other sources for specific test details.) (*continued*)

| Test | What It Detects | Basic Technique | Comments/Examples of Diseases Detected* |
|---|---|---|---|
| Hemagglutination (HA) | antibody | Soluble Ag is linked to RBCs and will agglutinate in the presence of Abs. | Example: Yersinia (plague). |
| Hemagglutination Inhibition Test (HI) | antibody | A standard virus suspension is added to serial dilutions of test serum and incubated. A standard RBC suspension is added. If Ab is present agglutination will be inhibited and RBCs will settle out as a button. The highest dilution of serum that inhibits hemagglutination is the HI titer. | Examples: bluetongue, African horse sickness, Yersinia. |
| Immunodiffusion | antigen or antibody | Ag and Ab are layered in a tube or placed in separate wells of agar (Agar Gel Immunodiffusion). Ag and Ab diffuse forming a visible precipitate when they meet. | Variations include radial and double immunodiffusion. Examples: anthrax, bluetongue, rinderpest. |
| Immunoperoxidase | antigen | Same as FA but uses enzyme-tagged (peroxidase) instead of fluorescein-tagged Ab. | |
| Latex Agglutination (LA) | antigen or antibody | Latex particles coated with Ag will agglutinate when mixed with the serum containing Ab. Particles can also be coated with Ab to detect Ag. | Example: Yersinia (plague). |
| Multiple-antigen print immunoassay (MAPIA) | antibody | A cocktail of antigens is applied by micro-aerosolization to a nitrocellulose membrane and incubated with test serum. The sample is washed and incubated with alkaline-phosphatase labeled anti-IgG antibody and enzyme substrate. Bound Ag-Ab complexes are detected by a measurable color change as in the ELISA. | Example: TB (experimental). |
| Nucleic acid hybridization | DNA or RNA sequences | Double-stranded nucleic acid is denatured and then applied to a nitrocellulose membrane or other support. A single-stranded DNA or RNA labeled probe (with nucleotide sequences specific to the target) is added. Complementary bases form bonds detectable by various assays. | The southern hybridization assay detects specific DNA fragments. The northern hybridization assay detects specific RNA sequences. Dot blot hybridization detects DNA or RNA. |
| Polymerase Chain Reaction (PCR) | specific viruses or specific gene sequences | An in vitro method of DNA replication. The test sample is heated to denature the DNA. Primers that are specific for the terminal ends of the target DNA are added. Repeated heating and cooling cycles in the presence of DNA polymerase enable the target sequence to be copied. The amplified target sequence can then be detected by electrophoresis and analyzed by measuring bands or by hybridization with specific probes. | Reverse transcriptase PCR used to test RNA samples. Examples: herpesvirus, TB. |
| Restriction Fragment Length Polymorphism (RFLP) | specific viruses or gene sequences | Restriction enzymes (endonucleases) are used to cleave DNA. The fragments are transferred to a membrane and probed to detect unique repetitive DNA sequences. | RFLP is based on mutations or genetic variability that exist at cleavage sites and is used to compare field isolates of particular organisms. Example: TB. |
| Serum Virus Neutralization (SVN) | antibody | Serial dilutions of test serum are incubated with a virus suspension containing 100–300 infective doses. Indicator cell cultures are added and cultures are observed for cytopathic effect (CPE). The presence of Ab will inhibit CPE. | Also used to identify unknown virus isolates by incubating with known Abs. Examples: herpesvirus, African horse sickness, bluetongue, rinderpest. |
| Western Blot Immunoassay | antigen | Test samples are loaded into a gel and Ag protein fragments are separated electrophoretically. The gel is transferred to a nitrocellulose membrane and then placed in a blocking solution to prevent nonspecific Ab binding to the nitrocellulose. The membrane is incubated with Ab, which binds specifically to the Ag and then with enzyme-conjugated Ab. Enzyme substrate is added, allowing visualization of Ab bound Ag fragments. | The Western Blot will confirm the presence of a particular microorganism and is often used to verify ELISA results. Examples: herpesvirus, TB. |

*Many of the diseases listed cause seroconversion but not disease in elephants. See Chapter 11.
References: 3,31,33,40,48,49.

our knowledge of heat balance in elephants[37,47] and provides a way to evaluate enclosures scientifically to determine appropriate shade structures for captive animals.[27,28,29,30] Thermography has not yet been widely used for clinical diagnosis in elephants. In one reported case, thermographic examination of a 45-year-old Asian elephant with trunk paralysis revealed lower temperatures than unaffected herdmates.[10] Elephants are affected by a number of problems that could benefit from thermographic evaluation. Other potential applications include evaluation of soft tissue injuries and foot problems. Although the expense of the equipment is prohibitive for most facilities, nearby veterinary schools may be resources for both equipment and expertise. It is likely that thermography will see increasing use in elephant medicine, and publication of case reports is encouraged.

## TUBERCULIN TESTING

Intradermal tuberculin testing is unreliable in elephants.[32,34] Diagnosis of TB by trunk wash (see the section "Culture," above) is preferred. Serological tests under study have shown promising preliminary results. Consult current Guidelines for the Control of Tuberculosis in Elephants (see Chapter 11).

## ULTRASONOGRAPHY

Basic ultrasound procedures are discussed in Chapter 27.

## URINALYSIS

Urinalysis is discussed in Chapter 29.

## VIRUS ISOLATION

Virus isolation is most successful during the acute phase of illness. Virus shedding diminishes as antibodies develop and virus is eliminated. The selection of clinical samples will depend on the disease suspected and/or the clinical signs that are presented. See Chapter 11. Viruses require living cells to replicate and must be grown on cell cultures. Virus may be detected by a characteristic cytopathic effect produced, stains that demonstrate viral inclusion bodies (e.g., herpesvirus) or fluorescent antibody that identifies viral antigen (e.g., rabies). Viruses can often be isolated from frozen tissue. In cases of acute death of unknown etiology, samples of major tissues (heart, liver, kidney, spleen) should be frozen routinely for possible virus isolation if histopathology does not identify the cause of death.

## WEIGHT

Knowing the weight of an elephant is vital for medicating, establishing appropriate feed intake, or the administration of chemical restraint agents. It is preferable to obtain weights by using scales, but if scales are not available, estimating weight to determine drug doses is the only choice. Scales for elephants are standard equipment in many zoological facilities in North America.

### Asian Elephants

A number of formulas are available for estimating weight in Asian elephants. These formulas have been mathematically derived from various body measurements, typically shoulder height and chest girth. None are absolutely precise for all age groups, and the degree to which the weight of an individual elephant may be over- or underestimated may be significant (≤ 80%). A given formula may appropriately be used in an individual elephant to monitor changes in weight over time. Selected formulas are presented in Table 13.4. In the author's experience, the method presented by Hile is both practical and subject to the least amount of error in adult elephants, but should not be used for juveniles.[18]

Recently an inexpensive instrument was produced by researchers in Sri Lanka to measure the height of ele-

**Table 13.4.** Selected Formulas to Estimate Body Weight in Asian Elephants

| Formula | Comments | Reference |
|---|---|---|
| Weight (kg) = 18.0 × HG (cm) − 3336<br>HG = heart girth circumference measured just caudal to elbow | N = 75<br>(8 males, 67 females; ages 1–57 years); adolescent age groups underrepresented<br>Average error ± 8%; not recommended for immature elephants | 18 |
| Weight (kg) = −1010 + 0.036 (L × G)<br>L = body length (cm) from the base of the forehead to the base of the tail<br>G = chest girth (cm) measured just caudal to the elbow. | N = 39 (30 males, 9 females; ages 15–50 years) | 41 |
| y = −22.39 + 18.9×<br>y = shoulder height (cm)[b]<br>x = cube root of the body weight (kg) | N = 39 (22 males, 17 females; ages 15–53 years); no calves included | 25 |
| y = −60.6 + 28.9×<br>y = chest girth (cm)[b]<br>x = cube root of the body weight (kg) | N = 39 (22 males, 17 females; ages 15–53 years); no calves included | 25 |

phants from a distance.[4] This instrument weighs only 4.5 kg and may be carried by one person. Two light rays and two crosshairs are aligned to the highest and lowest position of the elephant to calculate the height. The weight of the elephant is then estimated by a height-weight conversion of 30 cm = 227 kg (1 ft = 500 lb). The calculation appears to be approximately 70% accurate.

Some studies report that height is approximately twice the circumference of the pad;[25,26] however, other findings show this to be accurate only for immature elephants.[18] In the author's experience, foot circumference does not accurately predict shoulder height in Sumatran elephants, whose feet are typically broader than other Asian subspecies (poor nutrition among captive elephants may also play a role).

### African Elephants

Shoulder height and body length, expressed as logarithmic functions, correlate closely with body weight in African elephants.[24] In one study of 56 elephants, four measurements (chest girth, shoulder height, body length, and foot circumference), were all found to be valid estimates of body weight. Foot circumference was the most accurate, within ±6.0%.[7] A chart for veterinary use has been developed for estimating African elephant age and weight based on shoulder height.[6]

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# Section II: Radiology

Laurie Gage

## INTRODUCTION

Radiography of megavertebrates poses a set of unique technical challenges in acquiring quality diagnostic radiographs because of the massive size of the animals. Although adequate diagnostic techniques for radiography of the elephant foot and carpus have been employed for many years, it is only recently that newer technology allows more detailed radiographic evaluation of the limbs of elephants.[3] Diagnostic examinations of the abdomen and thorax as well as the spine, pelvic region, shoulder, head, and neck of the adult elephant are still limited by the available technology. These technical challenges have been met with relative success in horses, and in turn, equine techniques may be used on elephant calves.

Foot and limb disorders are one of the leading ailments of captive elephants.[6] Radiography has proven a useful diagnostic and prognostic tool for osteomyelitis in the elephant foot, as well as for diagnosis of arthritis or degenerative joint disease in the foot, carpus, or tarsus.[3,4] Radiography has also been used in many cases to identify or rule out deep infections to a damaged or cracked toenail or sole lesion. In one case, computer-enhanced radiographs were utilized in ruling out arthritis in the elbow joint of an adult elephant.[1] Because of the prevalence of foot disorders, survey radiographs of

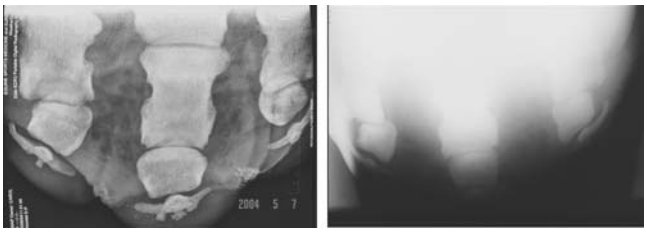

***Figure 13.3.*** *Left: Dorsopalmar view of the three central digits of the left front foot. This image was made using an Eklin digital radiography system. Right: Dorsopalmar view of the central digits of the left front foot using traditional radiography. Note, technique must be adjusted to optimize different phalanges.*

all the feet of each elephant would serve as a useful baseline to be included in every elephant's medical record.[2]

Newer technology has led to the improvement of the quality of radiographic images of the extremities of elephants. Digital radiology systems have improved the ability to produce detailed images of all of the bones of the foot, carpus, elbow, and tarsus. Digital radiography improves the ability to obtain diagnostic images of larger skeletal structures of the limbs as well. Digital radiography of the foot yields a more uniform image of all of the phalanges (Fig. 13.3, left), whereas traditional radiography requires multiple films of the foot at different techniques. A lower technique must be used to obtain diagnostic views of the fragile P3, whereas a higher technique is necessary to image the more dense P1 (Fig. 13.3, right).

Magnetic resonance imaging or MRI has been used to produce high-quality images of the structure of the distal limb in postmortem elephant limbs.

Computed tomography has been used in postmortem elephant limbs to pinpoint skeletal abnormalities, as well as to help define the anatomic orientation of the digits within the foot (Fig. 13.4).

## RADIOGRAPHIC EQUIPMENT

### Film/Screen Systems

Diagnostic films of the elephant foot may be obtained with a basic portable equine radiographic unit. The digits of the elephant foot are relatively easy to radiograph, provided the elephant is trained to offer its foot for the procedure.[3,5] A standard portable unit with 80 kVp and 15 mA with a film/cassette screen combination of 400 speed will be sufficient to obtain diagnostic films of the foot and possibly the carpus and tarsus.[3] Faster film/screen combinations combined with machines with higher kVp/mA capabilities allow more detailed studies of the long bones, carpus, tarsus, elbow, or portions of the stifle.

### Digital Radiography Systems

There are two types of digital radiography systems: computed radiography and digital radiography—both of which are becoming increasingly popular in veterinary practice.[7] Both digital radiography systems utilize the same type of radiographic unit to generate X-rays as the traditional radiography systems. Ideally the radiographic unit should be capable of generating 100 kVp and 20 mA.

Computed radiography (CR) utilizes a reusable, specialized phosphor-based plate housed in a cassette analogous to a film screen cassette. Once exposed, the whole cassette is placed into a reader that removes the imaging plate and reads it. After the image is read, the imaging plate is erased and placed back into the cassette. The image is sent to a computer and stored. This process takes approximately 90 seconds. When transferred to the computer, the image may be adjusted and manipulated. One image is obtained per cassette, requiring the use of multiple cassettes to perform a study. There is no time-saving benefit to this system over conventional radiography because one imaging plate is still required for each radiograph.

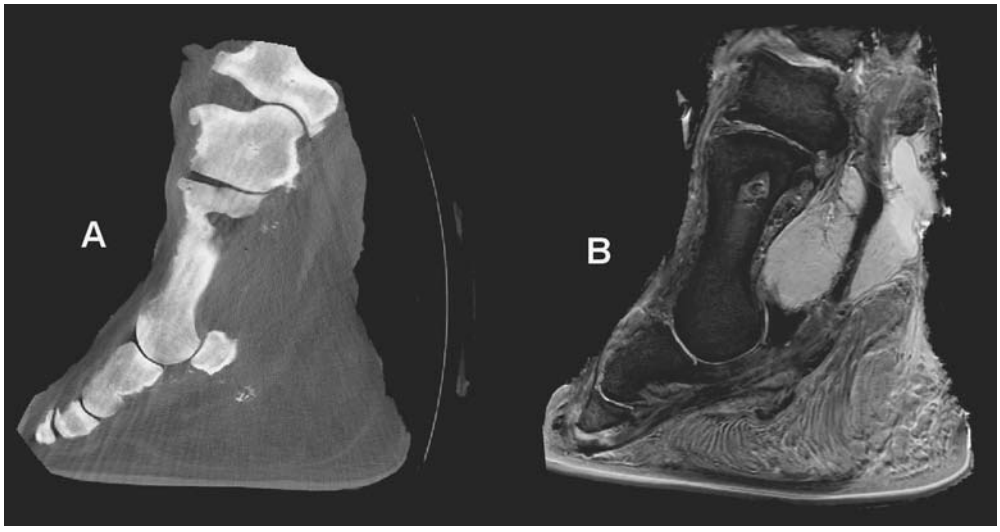


***Figure 13.4.*** *A) Postmortem CT Scan of an adult Asian elephant front foot. Note the proximity of the digits to the dorsal surface of the foot and the proximity of the distal phalanges (P2 and P3) to the toenail. (Image courtesy of Kimberly Luikart, DVM, and the UC Davis School of Veterinary Medicine Center for Imaging Sciences.) B) Postmortem MRI of an adult Asian female elephant front foot. Note the lesion in the metacarpal. (Image courtesy of Kimberly Luikart, DVM, and the UC Davis Imaging Research Center.)*

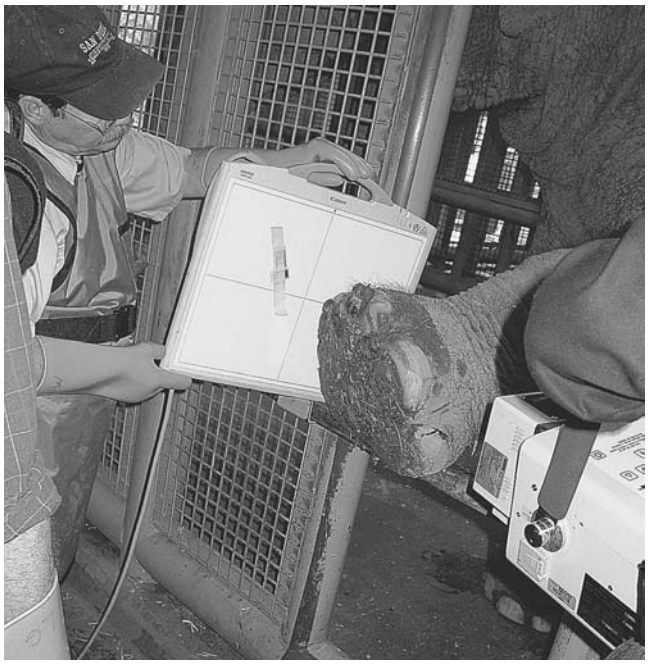

***Figure 13.5.*** *Obtaining a lateral view of the left front foot of an Asian elephant using the Eklin digital radiography system.*

Digital radiography (DR) employs a digital X-ray detector, similar in appearance to a standard X-ray plate; however, the portable large sensor panel has a handle (Fig. 13.5). The sensor panel is attached by a cable to a computer. The time from exposing the sensor panel to X-rays and viewing the image on the computer screen is 10 seconds or less.

Digital radiography equipment has the advantage of being able to produce a more detailed study of the structures of the elephant foot and leg using lower power techniques than those necessary for most film screen cassettes. The lower techniques and shorter times required to obtain images may allow diagnostic images of the larger bony structures, previously difficult, if not impossible, to achieve using film cassettes due to the density of the bones and thickness of the surrounding tissues. The DR sensor panel has the advantage of allowing different radiographic views to be performed in minutes by moving the radiographic unit without the need to reposition the elephant. The number of cassettes available does not limit a DR radiographic study; therefore, multiple-film studies may be accomplished in a fraction of the time it took when using film cassettes or the CR plates.

The disadvantages to the digital units are the high cost of the equipment and, in the case of the DR system, the need to have a computer in close proximity to the elephant because of its attachment to the sensor panel. A high-resolution monitor is necessary to read the images generated by either of the digital radiography systems.

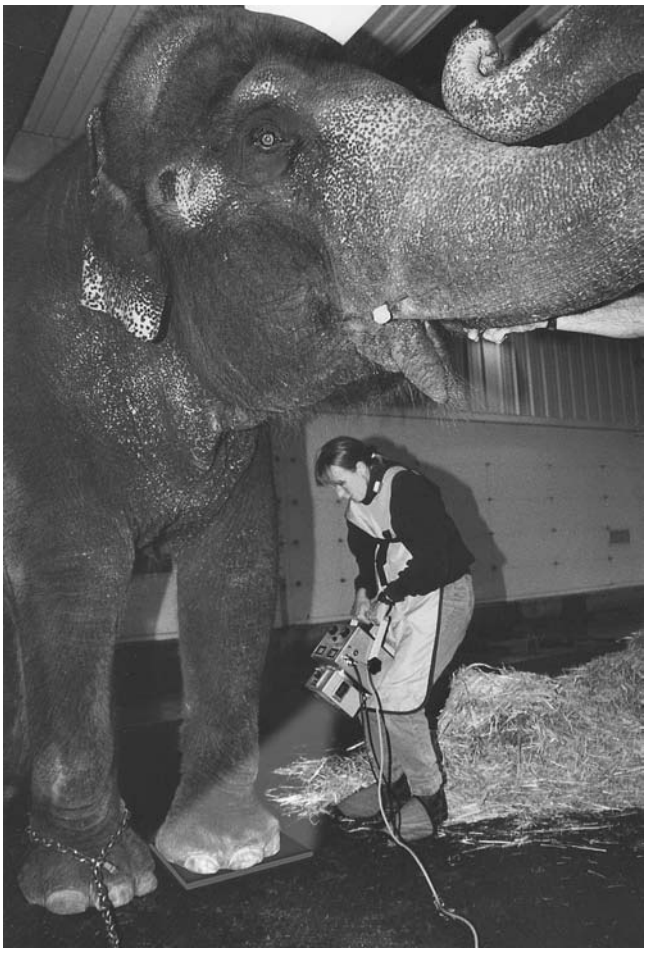

***Figure 13.6.*** *Positioning for a dorsopalmar view of the lateral digit of the left front foot. Note that the elephant is standing on a black film cassette. Note the lead shields covering the feet of the operator.*

## SAFETY CONSIDERATIONS

Elephants must be trained to carry out any radiographic study, whether the elephant is held in protected contact or free contact situations. Only experienced elephant training staff familiar with the individual elephant undergoing the radiographic study should handle that elephant. The veterinary radiology team should focus on the job at hand and work closely with the training staff when positioning the radiographic plates and radiology unit. Occasionally it may be prudent to allow the trainers to hold the radiographic cassettes. Everyone within 2–3 meters of the direction of the tube head should be wearing a lead apron. Anyone holding a cassette or cassette holder as well as the radiographic unit operator should be wearing leaded gloves. Radiographic unit operators should place lead blockers over their own feet to protect them from scatter when taking dorsopalmar views of elephant feet (Fig. 13.6).

**Table 13.5.** Estimated Times (in Seconds) Using a Radiographic Unit Set at 80 kVp and 15 mA and a 400 Speed Combination Film/Screen at a Distance of 60 cm

| View/Time | P3 | P2 | P1 | Metacarpus | Carpus |
|---|---|---|---|---|---|
| Dorsopalmar | 0.2–0.3 | 0.3–0.5 | 0.4–0.7 | 0. 5–0.8 | |
| Lateral | 0.3–0.4 | 0.5–0.7 | 0.6–0.9 | 0.7–1.2 | 4.0 |
| Anterior-posterior | | | | | 4.0 |

**Table 13.6.** Estimated Time (in Seconds) and Techniques Using a Digital Radiography X-ray Sensor Panel and Radiographic Unit Set at 100 kVp and 20 mA

| Position | P3, P2, Pl (all digits) | Metacarpal | Carpus | Tarsus |
|---|---|---|---|---|
| Dorsopalmar | 0.05–0.1 | 0.1–0.15 | n/a | n/a |
| Lateral | 0.1 | 0.15 | 0.25–0.3 | 0.25–0.3 |
| Anterior-posterior | | | 0.25–0.3 | 0.25–0.3 |

## RADIOGRAPHIC TECHNIQUE

Foot radiographs may be obtained using a standard equine portable unit. Because each x-ray unit and film cassette combination or digital radiography system differs, figures are offered as a starting point, but operators need to develop their own techniques for their equipment and their individual elephants. Techniques are listed in Tables 13.5 and 13.6 and were used to radiograph an average 3500 kg female Asian elephant.

Adjust techniques to compensate for the size of each animal. With a traditional film cassette system, the technique required to obtain detailed images of P1 is generally too high and yields an overexposed image of P3. Digital radiography allows a more uniform exposure of the entire foot without the need to adjust the technique to better image the individual phalanges (Fig. 13.3).

The thickness and corrugated character of the skin of the elephant must be considered when evaluating the images; because the skin of elephants is rough and wrinkled it tends to trap dirt and small particles (Fig. 13.7). The limb or foot should be washed or brushed thoroughly prior to performing a radiographic study. A stiff brush works well to clean the sole sufficiently for a radiographic study.

## POSITIONING THE RADIOGRAPHIC UNIT FOR VARIOUS STUDIES

Elephants are routinely trained to allow radiographic studies to be performed on their limbs and feet in both protected contact and free contact situations.[5] Quality radiographs are possible from both types of contact situations; however, due to cassette position challenges, obtaining certain images such as the elbow joint may be more easily accomplished with the animals trained for free contact.

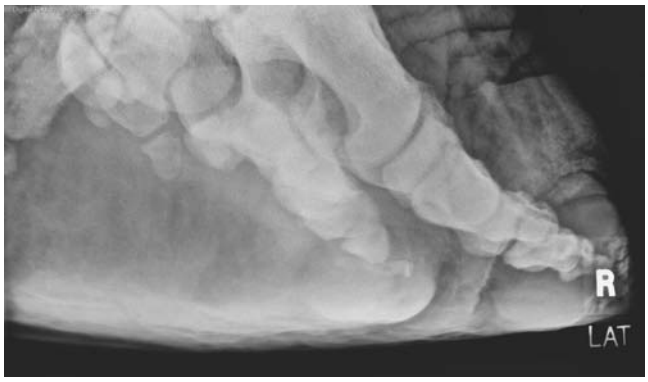


***Figure 13.7.*** *Lateral digital image of the right rear foot of an Asian elephant using the Eklin radiography system.*

The digits of the foot or the metacarpal bones may be evaluated by directing the beam of a portable radiographic unit in a dorsopalmar direction, at approximately a 50 ° angle, and holding the unit 55–60 cm from the film cassette (Fig. 13.6). Plastic 14 × 17 inch (35.6 × 43.2 cm), 400 combination speed Agfa film cassettes have been used with success for dozens of studies without damage to the cassette, in free contact situations. To obtain diagnostic images, the elephant is trained to place its foot directly on the cassette, which is positioned on a flat, clean floor. The sole of the foot should be free of dirt or stones. Any curvature of the floor will result in damage to the cassette. Protective wood cassette holders may also be constructed for the elephant to stand on.

Elephants also may be trained to present their limb over a sturdy bar, and various views of the foot may be obtained using creative cassette positioning (Fig. 13.5). These studies are most easily achieved using the DR sensor panels. This method of positioning the limb over a bar to obtain the various views may be preferable when using the expensive digital radiography panels, and it is commonly employed in protected contact situations. The elephant may also be trained to hold its leg in the correct position for other studies (Figs. 13.8, 13.9).

When osteomyelitis of the digits is suspected and a draining tract is present, it may be beneficial to instill an iodinated contrast agent such as Renografin into the tract to highlight it and determine whether it communicates with any of the phalanges. Depending on the depth of the tract, 1 to 3 ml of contrast agent may be injected directly into the tract and the opening plugged with some cotton or gauze. Radiographs of that digit should be taken immediately. See Figure 13.10.

Obtaining reasonably diagnostic films of the carpal joint with a standard 80 kVp 15 mA fixed setting radiographic unit has been accomplished by placing the

***Figure 13.8.*** *Positioning the rear limb of an Asian elephant for a posterior-anterior tarsal joint image.*

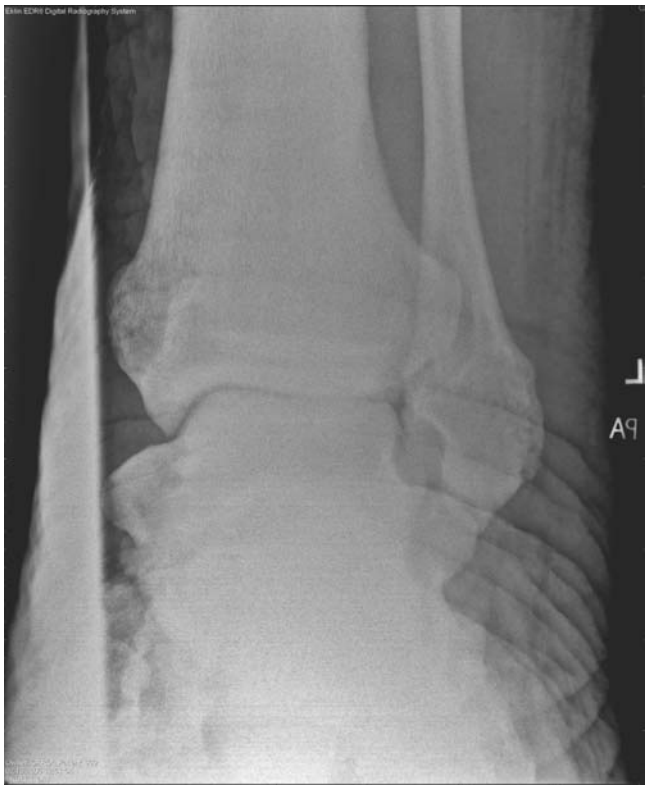


***Figure 13.9.*** *Posterior-anterior digital image of the left tarsal joint taken with the Eklin digital radiography system.*

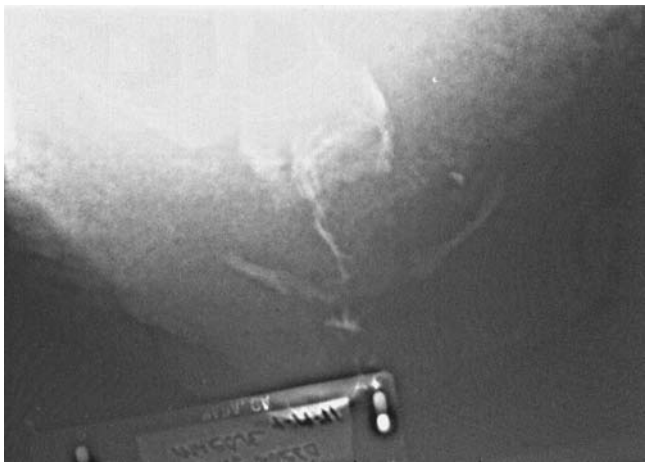

***Figure 13.10.*** *Contrast study of the draining tract of the lateral digit of the right front foot, dorsopalmar view. Note the extensive osteolysis of P3 and most of P2.*

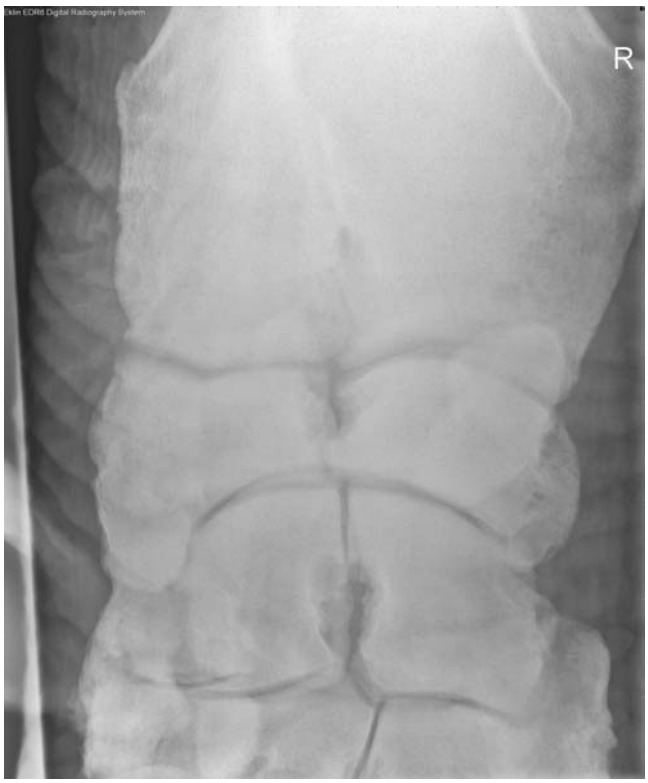


***Figure 13.11.*** *Digital AP image of the right carpal joint of a female Asian elephant using the Eklin digital radiography system.*

unit on a fixed stand, and using wide cloth tape to fix a 35.6 × 43.2 cm rare earth radiographic cassette to the carpal joint of the elephant. This procedure has been performed with free contact elephants.[3] Utilizing a stand for the radiographic unit and taping the cassette to the limb minimizes movement of the film or machine during the long exposure time. The elephant must be trained to allow this and be motionless for the 4 sec exposure to prevent blurring of the image. More powerful units combined with faster film/cassette speeds or the use of digital radiography produces more detailed films using shorter time settings. This allows the cassette to be held by hand or with a cassette holder without compromising the image (Fig. 13.11).

It is difficult to obtain detailed films of the elephant elbow using standard film cassettes; however, one study

produced reasonably diagnostic films with the use of computer enhancement of the images taken using a traditional radiographic system.[1] Digital radiography would likely allow better imaging of this joint. The cassette or sensor panel needs to be pressed into the axillary region as far anteriorly as possible between the elbow joint and the thorax, and a lateral or lateral oblique image may be obtained.

Posterior-anterior and lateral tarsal joints are imaged using both conventional film cassettes and digital radiography. For radiographic machines with less than 100 kVp capability, the radiography unit may need to be fixed to a stand due to the longer exposure time. The limb may be radiographed with the elephant in either a standing position or with the rear limb flexed at the stifle joint.

Due to the size and inaccessibility of the elephant stifle joint, diagnostic stifle images are difficult to obtain. The placement of the sensor plate or cassette is limited proximally and medially by the stifle skin fold. Diagnostic images, however, have been obtained using traditional radiography equipment when the elephant is cooperative.[4] Diagnostic images have been obtained using digital radiography using posterior-anterior positioning of the radiographic unit to the sensor panel. Digital radiography systems typically yield better images of this joint than do traditional radiography systems.

Diagnostic radiographs have been taken of the teeth and the jaws of elephants. The digital radiography systems seem to provide superior images of the jaws and teeth when compared to the images obtained using the film cassette systems.

## RESOURCES

The following are digital radiography resources used in this section:

http://www.eklin.com
http://www.gehealthcare.com

The following are computed radiography resources used in this section:

http:www.fujimed.com
http:www.idexx.com

## ACKNOWLEDGMENTS

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# 14 Postmortem Diagnostics

Richard J. Montali

## ELEPHANT NECROPSY PROCEDURES

Current knowledge of the actual performance of elephant necropsies is based mainly on early and somewhat fragmented literature of elephant anatomy and some later published dissections of adult[30] and fetal[11] Asian elephants. Much of this information has been summarized in a monograph on elephant medical management[17] and has served as the anatomical source of a brief necropsy procedural handbook emphasizing the Asian elephant.[2] Another field guide directed to the necropsy of the African elephant is also available[33] (http://www.elephantcare.org/protodoc_files/afrnecro.pdf).

The purpose of this chapter therefore will not be to duplicate the above guidelines; these should serve as adjunct resources prior to or during the necropsy. The scope will be to provide more of an overall strategy in the approach, initiation, and completion of postmortem examinations on elephants under captive conditions. This will include defining and illustrating pertinent unique anatomical differences in elephants important to achieve these aims, as well as some of the major diseases Asian and African elephants encounter in captive settings.

### General Plan

An elephant necropsy requires a concerted effort of 8 to 10 hours or more depending on its thoroughness and the resources and experience of the individuals conducting the examination. A number of organizations are currently available in the United States that may furnish elephant necropsy and research protocols and assistance with procedures of the dissection and sampling methods and tissue preservation. In some instances individuals and pathology teams with elephant clinical and pathology experience have been assembled in the U.S., and with proper notice may be made available to assist, particularly with any elective necropsies in elephants requiring euthanasia. The following documents are pertinent:

1. Elephant Necropsy Protocol (American Zoo and Aquarium Association [AZA], Elephant Species Survival Program [SSP])
2. Elephant Research and Tissue Request Protocol (AZA-SSP and the Elephant Research Foundation)
3. Guidelines for the Control of Tuberculosis in Elephants (The National Tuberculosis Working Group for Zoo and Wildlife Species)

Website addresses to obtain these and other elephant protocols are as follows:

www.aphis.usda.gov/ac/TBGuidelines2003.html
www.aphis.usda.gov/ac/ElephNecropsy2003.html
www.aazv.org
www.elephantcare.org

### General Approach

Elephant necropsies require a team approach with several pathologists, technicians, students, and lay assistants in attendance, if possible. Ideally, the necropsy should be performed where there is enough land to bury the elephant's remains by preparing a grave deep enough (at least 1.5–2 meters) for biohazard containment and preventing pets or wild animals from digging up the remains. The necropsy may be performed directly in the hole if it is big enough to contain the elephant and allow enough room for prosectors and assistants to maneuver. Obviously, these necropsy settings should be isolated from inhabited areas and well away from any possible underground public water or sewer services, and they may require authorized approval or a permit.

Transporting elephant carcasses requires heavy duty vehicles, including flatbed trucks, tractors with front-end loaders or cranes, and chain hoists. These are also useful to position the animal and for raising the limbs and moving major body areas as the dissection progresses. Vehicles must be able to handle the following approximate weights: female Asian elephant: 2,300–3,700

kg (5060–8140 lbs); male Asian elephant: 3,700–4,500 kg (8140–9900 lbs); female African elephant: 2,300–4,000 kg (5060–8800 lbs); male African elephant: 4,100–5,000 kg (9020–11,000 lbs). After the animal is positioned, smaller tractors can be used to lift limbs, rib cage, head, etc.

If transportation of the carcass or the necropsy procedure itself is delayed, the carcass may be covered with ice under a tarp (500–1000 lbs of ice may be piled on top of and/or beside the carcass to preserve tissues for up to 12 hours, even in summer heat).

If heavy equipment is not available it usually becomes necessary to perform the necropsy in situ using lighter equipment and/or a block and tackle (usually available at zoo or elephant facilities). The elephant carcass then must be manually reduced to convenient sizes for incineration, which requires a great deal of time and effort. All of this must be done with strict hygiene in mind to prevent the spread of infectious and known zoonotic diseases such as tuberculosis (TB)[12,18] and salmonella.[5] Humans and animals contacting the area used for elephant necropsies may be contaminated if the site is not properly disinfected. Disinfectants rated with high phenolic equivalence properties against mycobacteria should be used for surface contact decontamination. See Chapter 11.

Depending on availability, commercial rendering plants may pick up elephant carcasses deemed not to harbor infectious agents. Usually, they will only take muscle, bone, and viscera but not skin. Prior arrangements and conditions should be made. Further alternatives include commercial biohazard medical waste haulers, which may be expensive. New technologies that use sodium hydroxide under pressure are under study.

## Equipment Checklist

Recommended equipment and supplies are listed in Table 14.1.

**Table 14.1.** Equipment Checklist

1. Multiple scalpel handles, duplicates or triplicates of other instruments with extra boxes of scalpel blades, knife sharpeners (e.g., personal hand knife sharpeners, such as Accusharp, http://www.accusharp.com), steels, and a continual supply of new sharp knives.
2. Chain saw, axe, and/or reciprocating saw to cut through the cranium. A recommended model is the Wellsaw Model 404 (http://www.midwesternresearch.com/WELLSAW404.htm), hand and sledge hammers, chisels, and handsaws.
3. Standard large-animal necropsy instruments, including large tissue forceps to clamp off hollow viscous organs.
4. 4–6 hand meat hooks—very important for holding reflected skin and organs back while dissecting.
5. Sterile instruments for culture collection.
6. Field kits for acid-fast stains, gram stains, and rapid stains for cytology and blood cells.
7. Surgical masks approved to prevent TB exposure (example: 3M model N95). OSHA and CDC guidelines recommend at least N-,R-, or P-95 air-purifying facemask respirators or powered air-purifying respirators (PAPRs).
8. 10% neutral buffered formalin (2 gal); 4% buffered glutaraldehyde (100 ml) for electron microsocopy studies; Davidson's fixative for intact eyes (http://www.syndel.com/msds/davidsons_fixative_msds.html).
9. Formalin containers for sample collection.
10. Culture swabs, sterile urine cups, glass slides; serum tubes for blood and urine collection; aluminum foil and plastic bags for freezing tissues.
11. Liquid nitrogen or dry ice.
12. Labels and waterproof marking pens.
13. Scale for obtaining organ weights.
14. Tape measure (metric), at least 2 meters long.
15. Heavy duty electric hoist or crane; heavy duty ropes, chains with slip and grab hooks, and/or straps.
16. Wheelbarrow or carts on rollers to move heavy body parts.
17. Coveralls, boots, gloves, caps, masks, and protective eye and head gear.
18. Plastic biohazard containment bags and large heavy duty plastic barrels with sealable tops.
19. Accessible water supply with hose. (Caution! High velocity water equipment may aerosolize pathogens.)
20. Camera (conventional and/or digital) and film, extra batteries.
21. Tuberculocidal disinfectant for equipment; several are commercially available (tuberculocidal disinfectants should be specifically labeled to kill *M. tuberculosis*).
22. First aid kit.

## Special Considerations

Review the Elephant Necropsy Protocol (current version) prior to the necropsy and note disease issues that address specific tissue needs and special handling requirements; for example, in the U.S. and in some European countries TB (primarily caused by *Mycobacterium tuberculosis*),[15,16,20] and elephant endotheliotropic herpesviruses (EEHV)[6,21,26,28] have recently emerged. These and other pertinent diseases[14] are discussed later in this chapter. Research on these emerging diseases is ongoing. If the elephant is still alive, obtain whole blood and serum (~35–50 cc) for newly developed diagnostic tests for TB,[10] EEHV,[27] other research purposes, and banking. Blood may also be collected postmortem from the heart. If possible a final antemortem trunk lavage to detect mycobacteria should be performed (see Guidelines for the Control of Tuberculosis in Elephants). Also, review the Elephant Research and Tissue Request Protocol ahead of time. If time allows, contact researchers to confirm requests and verify collection methods and shipping arrangements.

## Necropsy Setup and Assignments

Set up a general necropsy area, with a "clean" place for collecting tissues for culturing and a larger space with 3 or 4 large cutting boards to examine organs and prepare tissue sections. Use neutral 10% buffered formalin preservation for light microscopy and 4% gluteraldehyde preservation for electron microscopy (EM). In addition, provide space for freezing (0°C) and ultrafreezing (−70°C) if liquid nitrogen or dry ice are available.

Assistants with knife-sharpening experience should be assigned to sharpen and cycle new knives continually to the primary prosectors. Several assistants should be assigned stations to receive bulk organs and to process them for culturing, preservation, or any special studies or handling as the necropsy progresses. If possible, weights and 3D measurements of major parenchymal organs (trimmed of fat and connective tissue) and brain should be recorded. In addition, a tally should be kept so organs or tissues are not overlooked or forgotten (See Table 14.2). These assistants should assure that all organs are grossly reviewed by the pathologist or other individual responsible for the case. An assistant should also coordinate the recording of all normal and pathologic observations and descriptions by pathologist(s) and other participating prosectors for the gross pathology documentation. Space should be provided for one more individual to photograph lesions and other findings.

A lead pathologist should be established to coordinate overall activities and, with one or two assistants, to initiate the necropsy. A great deal of effort must be made not to injure self or colleagues, particularly while removing the elephant's limbs and during the skinning process and entrance into the thoracic and abdominal cavities. After these initial procedures are accomplished, reassembly of the team or more prosectors, if available, can concentrate on different anatomical areas. While one group removes individual organs from the body cavities, another can perform more detailed dissections of the head in preparation for brain removal and of the limbs to view the joint surfaces, etc.

**Table 14.2.** Comprehensive Tissue Checklist

Preserve as many of the tissues listed below as possible in 10% buffered formalin at a ratio of approximately 1 part tissue to 10 parts solution. Tissues should be no thicker than 0.5–1.0 cm. For electron microscopy, fix diced (1 × 1 mm) pieces of kidney, liver, spleen, and lung in a suitable EM fixative such as glutaraldehyde. There is generally no need to fix and label each tissue separately. Take two sets of fixed tissue. Bank one set. Submit second set for histopathology.

Collect urine and abnormal fluids for cytology. Freeze postmortem serum (from heart), urine, and any abnormal fluid accumulations. Freeze 3–5 cm blocks of tissue from lesions and major organs (e.g., lung, liver, kidney, spleen) in small plastic bags. Freezing at −70°C in an ultralow freezer is preferred. If this is unavailable, freezing at conventional temperatures is acceptable (a freezer without an automatic defrost cycle is preferred). Consult the AZA Elephant Research and Tissue Request Protocol for specific research sample requests.

| | | | |
|---|---|---|---|
| Adrenal | Hemal node | Penis | Thymus |
| Blood | Kidney | Pituitary | Tongue |
| Bone with marrow | Large intestine | Prostate | Tonsillar lymphoid tissue |
| Bulbourethral gland | Liver | Salivary gland | Trachea |
| Brain | Lung | Skin | Trunk cross-section |
| Cecum | Parathyroid | Small intestine | Seminal vesicles |
| Diaphragm | Mammary gland | Spinal cord | Ureter |
| Epididymis | Muscle | Spleen | Urinary bladder |
| Esophagus | Nerve (sciatic) | Stomach | Vaginal/urogenital |
| Eye | Ovary/testis | Temporal gland | Uterus/cervix |
| Hepatic bile duct | Pancreas | Thyroid gland | |
| Heart/aorta | Lymph nodes* | | |

*Tracheobronchial, submandibular, tonsillar, mesenteric, axillary, inguinal.

## Order of Dissection

The order of organ observation, dissection, and removal should be systematic. As noted, since 1996, Asian elephants in the U.S. were found to have a >6% prevalence of pulmonary infections with *M. tuberculosis*.[15] Therefore, for safety measures, two methods of entering the thoracic cavity are presented: Method 1, for a low threat of TB, using a routine thoracic entry; and Method 2, for a known, or highly suspect, tuberculous elephant using a special thoracic entry to limit contamination of the necropsy site and team members' exposure to mycobacteria. In the latter case, the trunk should remain attached and clamped off after the postmortem trunk lavage is performed, and then it should be removed and dissected using Method 2.

To lower the risk of transmission of TB at that time, only the lead pathologist and one other prosector—with face masks (hepa-filter N-95, or PAPR), full face shields, disposable protective body garments, and durable gloves for hand protection against mycobacteria—should examine the upper respiratory and neck organs and the thoracic cavity, as will be described later. All personnel participating in or viewing the necropsy under conditions in which TB is suspected should have had a recent tuberculin skin test (within the previous 6 months) or radiographic examination for tuberculosis.

# DETAILED POSTMORTEM EXAMINATION

The Elephant Necropsy Protocol Gross Examination Worksheet may be used as a guide to document the inventory aspects of an elephant undergoing the necropsy. A condensed version of this worksheet is provided in Table 14.3. This should include important information as to the identity, origin, and ownership of each elephant case. It is extremely important also to have access to a detailed clinical history and/or events leading up to the elephant's death or reason(s) for elective euthanasia. Depending on the nature of the case, different levels of confidentiality and a secure chain of custody of samples and animal records may be required.

## External Examination

If possible, needed measurements should be taken and the skin should be examined when the elephant is still

**Table 14.3.** Elephant Gross Examination Checklist

1. General exam (physical and nutritional condition, skin, body orifices, superficial lymph nodes).
2. Musculoskeletal system (bones, marrow, joints, muscles).
3. Body cavities (fat stores, pleura, thymus, lymph nodes).
4. Spleen.
5. Respiratory system (trunk passages, pharynx, larynx, trachea, bronchi, lungs, regional lymph nodes; submit lung lesions for TB culture; bronchial lymph nodes should be cultured for TB even if normal in appearance).
6. Cardiovascular system (heart, pericardial sac, great vessels, myocardium, valves, chambers; be sure to examine abdominal aorta closely for subtle or obvious aneurysms).
7. Digestive system (mouth, teeth, tongue, esophagus, stomach, small intestine, cecum, large intestine, rectum, liver, pancreas, mesenteric lymph nodes).
8. Urinary system (kidneys, ureters, bladder, urethra).
9. Reproductive system (testes/ovaries, uterus and cervix, penis/vagina, urogenital canal, prostate, seminal vesicles, bulbourethral gland, mammary gland, placenta). Uterine masses/tumors are extremely common in Asian elephants, and multiple tumor types may be present.
10. Endocrine system (thyroids, parathyroids, adrenals, pituitary).
11. Central nervous system (brain, meninges, spinal cord).
12. Sensory organs (eyes, ears).

standing. Observe the overall nutritional condition of the animal by checking for prominence of head and hip bone protuberances for evidence of low body weight. Look for ventral edema, which occurs in captive elephants and has been associated with infection, diet, musculoskeletal disease, parasites, renal dysfunction, and an idiopathic transient form that resolves on its own.[14]

Before the carcass is finally placed in position, skin samples should be obtained from both sides of the animal when the carcass is being moved. A postmortem trunk lavage for TB should be obtained if not performed antemortem, and the trunk tip clamped if the elephant is a TB suspect. Any solitary or multiple skin lesions, observed on the elephant, should be taken for histologic and electron microscopic preservation and ultrafrozen for further diagnostic studies. Cutaneous papillomas (exophytic and inverted types) usually found around the trunk and face of younger wild and captive African elephants are the source of one of the EEHV viruses that can be fatal for Asian elephants.[9,28] See Figure 14.1.

Another potential source of this EEHV virus is the patches of lymphoid vulvitis (Fig. 14.2, Color Section) found in the mucosal surface of the distal urogenital canal in female African elephants.[23,28] In Asian elephants, hemorrhages and cyanosis of the tongue (Fig. 14.3A, Color Section), peri-oral muco-cutaneous and palatine ulcers, (Fig. 14.3B, color plate), and trunk and front leg edema can be typical signs of acute fatal EEHV infection.[28] Another EEHV virus also carried by African elephants but in pulmonary lymphoid nodules,[13] has been attributed to several cases of fatal disease in captive African elephants[28] similar to that of Asian elephants.

In Europe, rodent-borne elephant pox (bovine orthopoxvirus) (Fig. 14.4, Color Section), more common and severe in Asian elephants than in African elephants, may present with large ulcerated lesions, classically on the face and feet, but also anywhere else on the body.[24] Elephant pox is a zoonotic disease in Europe.[18]

The external examination should also include the eyes, which have a prominent nictitating membrane with a large, lobular brown Harderian-like gland[11] but no functional lacrimal or orbital glands. Examine also the ears, mucous membranes, all body orifices, external genitalia, and feet.

Unique structures in elephants are paired temporal (musth) glands (Fig. 14.5, Color Section) that are located in the temporal depressions between the orbital fossa and external ear canal. The temporal glands have a major duct opening that allows drainage of an apocrine secretion down the side of the face. These glands are larger in bull elephants that are sexually active. Unique "external" genitalia include internalized testes in the retroperitoneum in bulls. A long urogenital canal with an orifice that opens low and cranial to the hindlegs, as well as paired pectoral mammary glands, each with a nipple, occur in cow elephants. Numbers of digits (five) are constant in the front and back feet and do not always correlate with numbers of toenails, which may be reduced in both front and back feet in Asian elephants and the two species of African elephants.[11,25]

The proboscis or trunk differs at the distal end, with Asian elephants having one prehensile finger-like tip and the African elephant with two similar finger-like tips. If the elephant is a TB suspect and the trunk has been clamped, it should await Method 2 before examining respiratory tract organs. If not, the trunk may be transected close to the head and both of the separate nasal passages slit longitudinally and sampled. Trunk paresis and paralysis (floppy trunk syndrome) has been described in wild African elephants as outbreaks, often in bulls, possibly associated with plant or environmental toxins. In captive elephants most trunk mobility problems are sporadic and injury related, as summarized,[14,17] although a trunk paresis of unknown etiology has been reported anecdotally in captive Asian elephants in North America.

## Carcass Positioning and Limb Removal

If heavy-duty equipment is available, the elephant should be placed on its left side. The right thoracic limb is removed while being lifted by the chain hoist (Fig. 14.6) or by pulling the limbs dorsolaterally with a tractor positioned behind the spine of the animal. Limb removal requires deep incisions into the axillary skin and dorsally around the skin and muscles of the scapula. Likewise remove the upper-right pelvic limb after disarticulating at the hip (note the lack of a round ligament in the femoral head[11]) and cutting through the dorsal

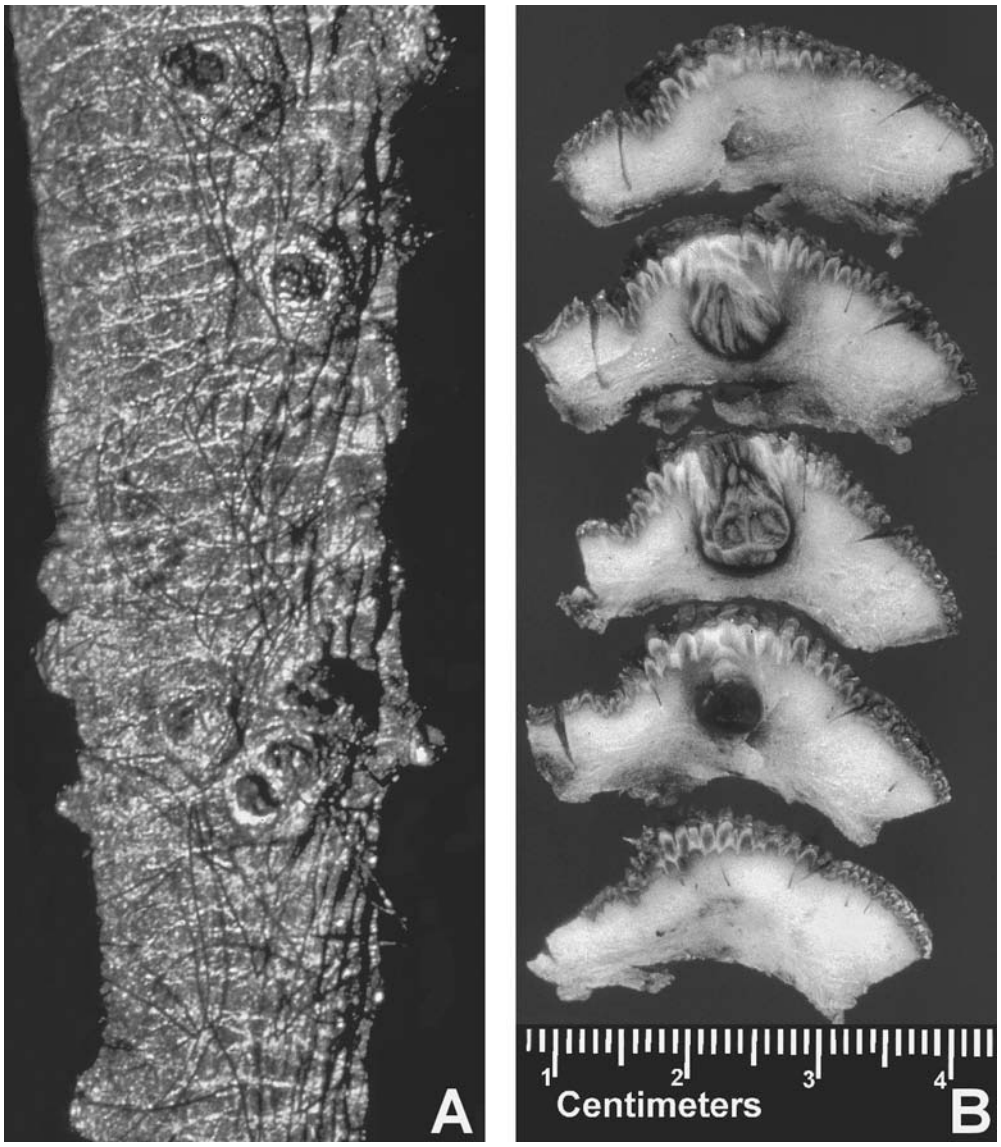


***Figure 14.1.*** *Cutaneous inverted papillomas from captive African elephants that harbor elephant endotheliotropic herpesvirus (EEHV) may be fatal for Asian elephants. A) Trunk lesions, B) sectioned trunk lesions illustrating inverted growth of papillomas (courtesy of Dr. E. Jacobson and JAVMA*

hip skin and musculature. (Eventually the left thoracic and pelvic limbs may be removed similarly after the body cavities are examined and eviscerated, and all the limbs may be dissected.)

After removing the upper thoracic and pelvic limbs, continue skinning over the upper-right lateral side to the dorsal midline and cranial to cervical and mandibular regions. Meat hooks or a hoisted chain may be "threaded" into transcutaneous incisions into the skin, and large segments of skin may be lifted and cut away. Reflect the lower abdominal skin downward. Examine and sample skin, temporal gland, peripheral lymph nodes (axillary, inguinal, mandibular, prescapular, but no popliteal noted in African elephants[33]), and salivary glands (parotid, submaxillary, and sublingual).

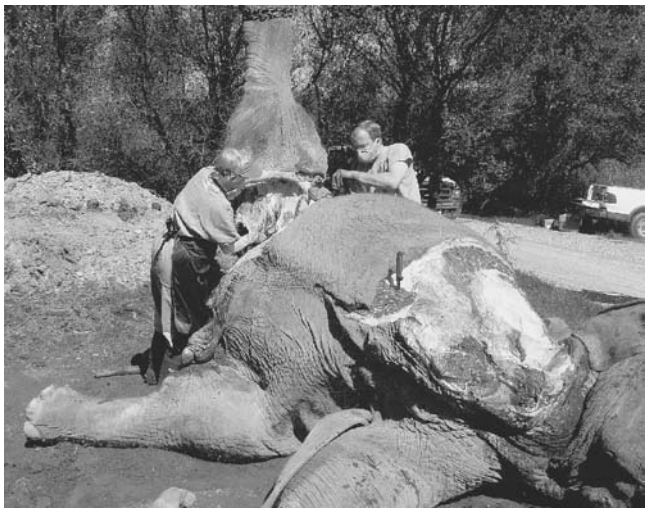

***Figure 14.6.*** *Carcass positioning and upper limb removal of an adult female Asian elephant for necropsy. Example of using a crane in an outdoor setting.*

## Abdominal Cavity

With a reasonably sharp, curved knife or large scalpel with a heavy duty blade, carefully make a midline inci-

sion from xiphoid to pubis using multiple light strokes until through the peritoneum; repel the intestines as you remove the upper and lower body wall. Note the amount of abdominal fat storage to assess nutritional condition. In young elephants, numerous petechial hemorrhages uniformly throughout the mesentery and serosal surfaces are a sign of EEHV[28] (Figs. 14.7A, B, Color Section).

The hollow visci are massive and require at least two other assistants to remove them intact effectively and dissect them. The greater omentum wraps around the stomach and small intestine, and it normally has low fat content.[30] Cut the greater omentum at its attachment from the greater curvature of the large, saccular, equine-like stomach, and remove the elongated spleen and slice it into narrow segments. Identify the duodenum at its U-shaped loop and find the lobular pancreas in the mesoduodenum. The hepatic bile duct and major pancreatic duct enter the lateral side of the duodenum at a point with numerous mucosal folds (ampulla).[11] Transect the ducts; the bile duct may have calculi in older elephants[33] (and unpublished data, R. J. Montali). Take samples of the pancreas at several levels and from the ducts.

With large forceps, double-clamp the duodenum and transect it below the pylorus. Likewise transect the rectum high in the pelvis. With assistance manipulating the organs, use blunt and semisharp dissection down to the root of the mesentery to detach the small and large intestines proper and cecum from the abdominal cavity. Transect the esophagus at the cardia and remove the stomach and liver separately.

Open the stomach on its greater curvature and then the entire length of the intestinal tract. Take special note of the gastric mucosa because erosions and ulcers are common in animals that have received long-term nonsteroidal or steroidal antiinflammatory therapy. Identify mesenteric lymph nodes and take lymph node and intestinal cultures and samples for preservation. Salmonella is an important pathogen in captive elephants.[5] The cecum has an ileocecal orifice. Sand is a common finding in the cecal lumen (1.2 kg in one case[30] and 1–2 kg in another case) (unpublished data, R. J. Montali). The liver has two lobes, of which the right lobe is larger and subdivided.[3] A gallbladder is not present in African or Asian elephant species. Slice the liver into narrow segments and take samples, as well as from all gastrointestinal segments, for preservation in neutral 10% buffered formalin.

The adrenals, kidneys, and testes are located in the retroperitoneum in the thoracolumbar spaces. The adrenal glands are not readily visible in the retroperitoneum. Palpate for them anterior and ventral to the kidneys; they have a distinctive firm texture. Define and dissect the adrenals away from the kidney. In the Asian elephant, the left adrenal is smaller than the right and pyramidal in shape.[11] The testes lie caudal and ventral to the kidneys at about midlumbar level; a complex of coiled ductular structures replaces a true epididymis in the elephant.

The kidneys are lobular with multiple renal papillae (multipyramidal); the left kidney is smaller than the right. The urinary bladder has a potential volume of 18 l when filled with water.[30] However, the physiological capacity during life has been noted to be 4–6 l.[8] Section and sample all of these organs. Renal disease occurs infrequently in elephants.[14,17] A recent report of pyelonephritis in an Asian elephant provides important updated clinicopathological information about urinary tract infections in Asian elephants.[29]

The remainder of the male urogenital tract as summarized[17] includes paired thick-walled seminal vesicles, a prostate located on the dorsal aspect of the urethra, and large bulbourethral (Cowper's) glands; samples from all these should be taken for formalin fixation.

The female elephant reproductive tract as summarized[17] is bicornuate with the two horns attached just before the body, a cervix, flat lobulated ovaries with bursae, and prominent fimbria of the oviduct. The lower tract is unique in both Asian and African elephants and is characterized by a long urogenital canal (80–100 cm or longer) that originates at the pelvic brim and ends at the vulva anterior between the back legs.

The female genital tract may be removed in two sections as follows: the anterior segment with part of the uterine body, horns, and the ovaries and urinary bladder by transecting the uterine body midway in the pelvis and mesenteric attachments of the remaining anterior segments and delivering through the inguinum from the abdominal cavity side. The caudal segment may be removed with the urogenital canal by dissecting ventral to the coccygeal vertebrae at the head of the tail and around the pelvic outlet and inlet. Delivering the entire urogenital tract in toto is also possible, but it requires more effort dissecting. Samples from all segments of the urogenital tract should be taken for diagnostic and research purposes.

Important findings in recent pathologic studies of the female elephant reproductive tracts are as follows: Asian but also African elephants have a propensity for uterine cystic and polypoid endometrial hyperplasia (Fig. 14.8, Color Section). There is a high prevalence in nulliparous cows over 26 years of age,[1] and fertility may be affected. In addition, nulliparous Asian elephants usually older than 20 are predisposed to uterine leiomyomas[19] (Figs. 14.9A, B, Color Section), some of which may be massive, weighing more than 100 kg (unpublished data, S. Terrell), with several undocumented cases of leiomyosarcoma in old cows (unpublished data, R. J. Montali). Uterine leiomyomas do not occur as a problem in African elephants.[8] However, polyps in the urogenital canal of African elephants[22] (and unpublished data, R. J. Montali) have been observed, as well as lymphocytic vulvitis in both Asian and African species.[23]

EEHV has been identified by PCR in the lymphoid vulvitis lesions in African elephants (Fig. 14.2, color plate) but not in Asian elephants.[28]

## Thoracic Cavity

**Method 1 (no threat of TB).** Elephants have 7 cervical vertebrae, 19–21 thoracic vertebrae, usually 19 pairs of ribs (that might vary with the numbers of thoracic vertebrae) and 3–5 lumbar vertebrae. Some variations occur between African and Asian elephants, mainly with the vertebrae.[11,30] Cut the upper (right) ribs near their vertebral and costo-chondral attachments at right angles from anterior to posterior with an oscillating or chain saw. With a knife, cut a small tunnel through the ventral (parietal) surface of one of the foremost anterior ribs near the thoracic inlet and encircle a chain around it. Then carefully cut the intercostal muscles and the dense pleural connective tissue under the ventral rib surfaces as the ribs are lifted with a hoist. Remove the upper rib cage in two or three sections at a time. Be careful not to cut into the lung tissue because elephants are devoid of a pleural space due to parietal and visceral pleural fusion.[11,30]

Dissect and deliver the tongue through the intermandibular space and the neck organs to the level of the thoracic inlet. The thyroid gland is bilobed with a thin isthmus and lies ventrolaterally within the first four rings of the proximal trachea.[11] Up to two pairs of parathyroids may be found[33] (but not easily) in the ventral edge of the thyroid lobes.

The thoracic organs with the tongue and trachea may then be freed by cutting the remaining pulmonary parietal–pleural connections and dense dorsal and mediastinal ligamentous attachments to the pericardium at the base of the heart.

Examine the tongue, tonsillar crypts, and tonsils (histologically proven to exist in adult Asian elephant—unpublished data, Linda Lowenstine) larynx, tracheal and esophageal mucosa, and major bronchi, and take samples. The right lung is larger than the left; a prominent cardiac notch is present and both right and left lung lobes have foreshortened lobular separations.[11,30] At this point bronchial lymph node samples (fresh frozen or ultrafrozen) should be taken for mycobacterial cultures even if there is no gross evidence of tuberculosis. Also preserve lung sections and regional lymph nodes (in buffered 10% neutral formalin as recommended in The Elephant Necropsy Protocol).

The elephant heart is globoid and weighs up to 0.5% of the animal's total body weight. The heart usually has distinctive left and right ventricular apices referred to as a *bifid apex*.[11,30] There are paired anterior vena cavae, and the ductus arteriosus joins the aortic arch with the left branch of the pulmonary artery[11] rather than the pulmonary arterial trunk as in most ungulates. In North America, the elephant heart is the target of two fatal viral diseases, encephalomyocarditis virus (EMC)[7,32] and the previously mentioned EEHV. Gross findings of EMC are pale streaks and/or epicardial hemorrhages[32] (Fig. 14.10, Color Section). Cardiac hemorrhages from EEHV are usually extensive, with involvement of the epimyocardium and endocardium[28] (Figs. 14.11A, B, Color Section). Arteriosclerosis, mostly in the form of medial calcification and uncomplicated intimal atheromatous–type lesions are described mainly in wild African elephants and are believed to be associated with environmental stressors.[31]

**Method 2 (threat of TB).** If there is antemortem evidence of TB, a reasonable suspicion of TB, or absence of any TB testing history, entrance into the thoracic cavity by the lead pathologist and an assistant can be approached from the intact diaphragm after all abdominal viscera have been removed. All other necropsy participants should be dismissed from the area before the thoracic cavity is opened, and should not return until the thoracic and upper respiratory organs are cleared of having acid-fast bacilli.

The diaphragm may now be cut from its costosternal attachments. Typically, the earliest lesions of TB would be found in the lungs (Fig. 14.12A, Color Section) and bronchial lymph nodes, which can be palpated from a caudal approach as the lobes are being separated from the closely joined visceral and parietal pleura. However, massive involvement of the lungs with caseocalcareous and cavitary lesions are not uncommon in elephants with prolonged, advancing tuberculosis (Figs. 14.13A, B, Color Section). A good reason for postmortem clamping of the trunk tip in a known tuberculous elephant is shown in Figure 14.14.

Dissect and deliver the tongue through the inter-

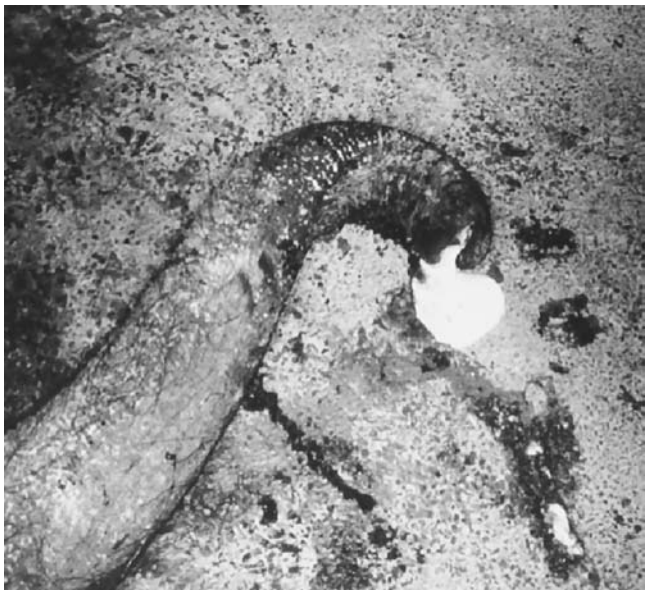

***Figure 14.14.*** *Tuberculous exudate draining from the trunk tip. The lung from this elephant is depicted in Figure 14.13B (color plate) (courtesy of Dr. H. Kinde).*

mandibular space, as described in the second paragraph of Method 1, and examine and sample neck organs as noted. A careful examination should be made of the freed-up neck organs and clamped-off trunk for tuberculous tonsillar lesions and laryngo-tracheal (Fig. 14.12B, color plate) or trunk mucosal plaques. These lesions may occur in both early and late stages of tuberculosis and are likely be a shedding source of TB organisms.[20]

Acid-fast stains should be performed on any suspicious lesions immediately. If found to be positive, and depending on the degree of involvement, the thoracic organs should continue to be examined but less invasively. Samples should be quickly collected to achieve a specific mycobacterial diagnosis. The bulk of the thoracic organs should then be treated as biohazard waste by deep burial or by using special containment plastic bags and buckets. After mycobacterial infection is confirmed (or still strongly suspected), power saws and chain saws should be used either on the carcass with extra precaution or not at all to prevent the aerosolization of mycobacteria. Extrapulmonary sites of tuberculosis should also be evaluated, including bones and joints.

At that point team participants returning to the necropsy should also take further precautions against potential mycobacteria contact or have the option to withdraw from the remaining necropsy. OSHA standards (29CFR1910.134) require that "workers present during the performance of high hazard procedures on individuals (humans) with suspicious or confirmed TB" be given access to protective respirators (at least N-95 level masks). Similar precautions should be taken during an elephant necropsy that involves risks for exposure to TB. If there is no evidence of tuberculosis, the heart, lungs, and associated structures may then be removed en bloc, as previously described, and further dissected as in Method 1.

## Dissection of Limbs

The joints of the lower limbs, particularly the front feet and hindfeet, are complicated, and thorough examinations cannot be performed within the 8–10 hours previously quoted for a complete elephant necropsy. Good anatomical correlations of bone and soft parts of the limbs may be held for perfusion fixation with neutral buffered formalin or frozen for radiographic, computerized tomography or MRI imaging for subsequent in-depth study. However, the carpal, shoulder, tarsal, stifle, and hip joints are more approachable, and joint fluid and articular surfaces should be examined and samples taken for histology. Some of these joints may be approached more easily by suspending detached limbs from a hoist or tractor and using the weight of the distal limb to open the joint after dissection (personal communication, S. Terrell). Elephants are prone to degenerative osteoarthritis[14,17] and inflammatory joint disease associated with *Mycoplasma* spp.[14]

## Dissection of the Head

The caudal segment of the head should be skinned and muscle dissected away to the level of the caudal edge of the orbits. If not previously done, remove both eyes by dissection with a long-handled scalpel and place whole without injecting in Davidson's fixative (http://www.syndel.com/msds/davidsons_fixative_msds.html) for best preservation. With a heavy-duty, straight-pointed knife, loosen the attachments between the occiput and the first cervical vertebra. Attempt to expose the dorsal aspect of the foramen magnum and transect the spinal cord.

Continue to separate the head at the atlanto-occipital junction by allowing the knife blade to find its way between the joints on both sides of the condyles while moving the head—or having the head moved by an assistant—side to side and up and down. Removal of the brain may be accomplished by several methods as follows:

**Method 1.** A caudal approach to the brain removal may be made by three connecting deep inward cuts with the curved end of a chain saw parallel along the margins of the flattened triangle formed at the base of the elephant skull. The left and right lines begin at the medial edges of the condyles at the foramen magnum remaining parallel to the left and right outer margins of the flattened skull and connect with the dorsal horizontal line on each side. Then carefully remove the surface and the deep cancellous bony segments (these contain prominent air sinuses in the elephant skull bones that increase with age[4]) overlying the anterior cranium with a curved crowbar. When the margins of the brain are visualized, further careful removal of bone fragments with hand instruments should enable the entire brain to be delivered caudally. See brain removal Method 2 below for pituitary removal and brain preservation.

Caution! Use of a chain saw on bone may be hazardous and cause shrapnel-like fragments to be launched. Protective eye, head, and face gear should be worn by the chain saw operator and any personnel in the immediate area.

**Method 2. (Dr. Dalen Agnew, unpublished data).** The following procedure is more detailed, but it results in a relatively intact skull, and the brain itself is not damaged, though it is removed in two parts. Required instruments include a scalpel or small necropsy knife and a carpenter's electric reciprocating saw (Milwaukee Sawzall® or similar) with a supply of 23 cm (9 in) wood/nail cutting blades. A hand keyhole saw or jigsaw may be used in the field if electrical power is unavailable. An ax or hatchet may also be used, but the brain may be damaged and the physical effort required is much greater.

With the elephant in lateral recumbency, remove all the skin from the neck to the eyes and over the top of the head. Then follow these steps:

1. Cut down to the atlanto-occipital junction from the dorsum. This may be done by working ventrally along the back of the skull to the spinal cord. When the atlanto-occipital joint is found, the head may be rolled forward slightly to open the space between the atlas and the occipital condyles. Drive the sharp knife into this space to sever the spinal cord. (This prevents further histologic stretch artifacts in the brainstem and spinal cord.)
2. Dissect all the muscle from the caudal aspect of the skull so that only bone is exposed approximately 15 cm dorsal and 10 cm lateral to the foramen magnum.
3. With the power saw, make the following cuts (Fig. 14.15):
   a. From the ventral lateral aspect of the foramen magnum, cut dorsally 15 cm at a 30° angle from the ventral midline on both sides (a in the figure).
   b. Connect the dorsal extent of both cuts with a third cut. In order to cut through the bone, it is best to lay the saw flat on the surface of the bone until it cuts through to the cranium. Then the cut may be extended laterally to connect with the first two cuts (b in the figure).
   c. The bone fragment may now be removed. A pry bar may be used if necessary to separate the bone if the cuts are not complete.
   d. If the resulting widened foramen magnum is not sufficiently separated to remove the brain, two additional cuts may be used to increase the width. Cut laterally from the base of the foramen magnum approximately 8 cm; then cut from the most cranial aspect of the skull ventrally to the end of the previous lateral cuts (d in figure).
4. The cerebellum and brainstem are now exposed. Reach in with the knife or a scalpel and separate the cerebellum and brainstem from the cerebrum. The cerebellum may be now be removed carefully, cutting cranial nerves as it is elevated.
5. The cerebrum may be extracted now by reaching in with a scalpel or simply a gloved hand, carefully separating the brain from its dural attachments and delivering it through the widened foramen magnum.
6. Usually the pituitary gland remains in situ and may be removed by carefully lifting it by the dural attachments with forceps and using a scalpel to peel it from the underlying bone.

The brain should be preserved separately in 10% neutral buffered formalin at a 10:1 ratio of formalin:brain. In the first method of removing the brain in toto, it is best to make a sagittal incision into the corpus callosum to allow the formalin to perfuse the ventricles. If gross morphology of the brain is not critical, the second method allows separating cerebral hemispheres sagittally and preserving cerebral segments and the cerebellum separately.

When the vomeronasal organ needs to be removed it is important to preserve, as much as possible, the anterior olfactory segment of the brain with some of the cribiform plate intact and the palatine openings. For more details, consult the Elephant Research and Tissue Request Protocol if that is required.

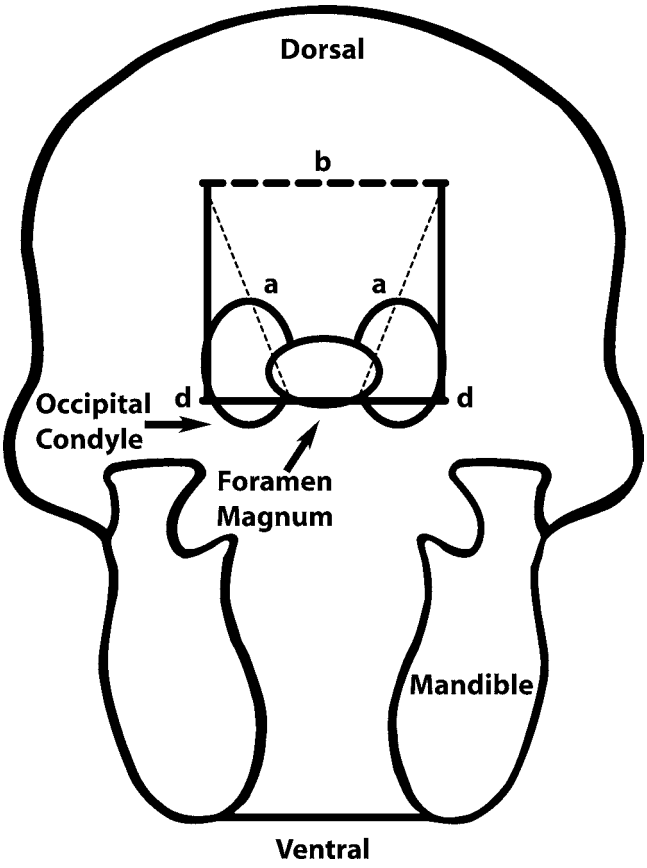


***Figure 14.15.*** *Graphic sketch of posterior aspect of an elephant skull illustrating landmarks for brain removal (courtesy of Dr. D. Agnew).*

It is important to ultrafreeze brain tissue for anticipated diagnostic purposes, such as isolation of novel viral and other infectious agents, or for genetic research purposes. Therefore, prior to fixation, cut small ($1 \times 2$ cm) block sections from the cerebrum, cerebellum, and medulla oblongata for ultrafreezing in dry ice or liquid nitrogen for transporting to an ultrafreezer at $-70$°C or lower.

For spinal cord samples, take three or four 10 cm sections of vertebrae (C1-C2, C5-C6, T6-T7, L2-L3, for example) with the reciprocating Wellsaw and then "fish out" the spinal cord segments using a long-handled scalpel and fix in 10% buffered formalin in divided sections (unpublished information, Dalen Agnew).

## CONCLUSION

Performing relatively detailed postmortem examinations on elephants is a laborious task requiring many

hours and major resources; however, these exercises are integral to comprehending and solving disease problems. Some of the most serious infectious diseases have recently emerged in elephants.[15,27] New imaging techniques correlated with pathologic findings have opened the way to the diagnosis of reproductive tract diseases that have hampered fertility in captive elephant populations.[8,19]

It remains very important then to continue to expand the medical knowledge base by gaining new pathology information. A great deal of this may be obtained by continuing more directed and detailed postmortem examinations in any elephant that dies or has to be euthanized. It is the hope that this chapter, calling for a systematic approach with cooperation and teamwork, will help provide a more useful means to those aims.

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# 15 Therapeutics

Susan K. Mikota

## INTRODUCTION

Our collective view of the pathophysiology, diagnosis, and treatment of disease in elephants continues to evolve as we gain knowledge of health and disease on levels that range from molecular to ecological. The complex environmental issues that may impact the health of wild elephant populations are briefly discussed in other chapters of this book (see Chapters 7 and 24).

This chapter presents methods used to administer medications to individual elephants and includes a compilation of pharmaceutical products. It is by no means comprehensive, and documentation of drugs used in every country is beyond the present scope. Major drug groups are described, and published information pertaining to elephants is cited. An Elephant Formulary that is updated periodically by the author can be found online at http://www.elephantcare.org/drugdos.htm.

Few drugs used in elephants have been subjected to pharmacokinetic analysis, and most dosages are anecdotal or extrapolated from other domestic species (mainly the horse).

> We treat animals about which we know little,
> for diseases about which we know less, with drugs
> about which we know nothing at all."
>
> Anon.

The lack of scientific research in this area is astounding considering the endangered status of this species and the attention given to other aspects of its care. Further research is needed.

## ABBREVIATIONS USED IN THIS CHAPTER

CNS: central nervous system
CSF: cerebrospinal fluid
GI: gastrointestinal
h: hour
IM: intramuscular
IV: intravenous
PO: orally
pk: pharmacokinetics
q 6h: administration every 6 hours (QID)
q 8h: administration every 8 hours (TID)
q 12h: administration every 12 hours (BID)
q 24h: administration every 24 hours (SID)
SQ: subcutaneous

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## DETERMINING DRUG DOSAGES

There are five components to a dosage regimen: the formulation, the dose, the route, the dosing interval, and the duration of treatment. Pharmacokinetics (pk) is the study of drug movement through the body (absorption, distribution in various tissues or fluids, biotransformation, and elimination). Pharmacokinetic studies determine dosage regimens and ideally are performed for each new target species. Only a few such studies have been conducted in elephants, generally with small sample sizes that have precluded determining differences between species, age groups, or between captive and free-ranging elephants. Where elephant-specific information is lacking, extrapolation from the equine dosages is commonly used and provides a logical guideline until further elephant studies become available.

Given the plethora of variables already present, it is essential to begin with an accurate weight if at all possible. A measured (scale) weight is preferable but estimates may be used if necessary. Refer to Chapter 13, for information on estimating weight. After a drug and dosage have been selected, the challenging task of administra-

tion remains. This is discussed below. As a routine practice, drugs should not be mixed because many are physically incompatible.

### Metabolic (Allometric) Scaling

Measurable biological parameters (e.g., oxygen consumption, cardiac output) demonstrate a logarithmic, linear relationship with body weight. This direct relationship between metabolic rate and mass exists across five major taxa and is the basis for the metabolic scaling of drug dosages. Numerous articles have been written on the topic.[3,4,38,59,60] The basic concept is to use a known dose of a drug for a domestic species (in this case, the horse) to calculate an extrapolated dose for the elephant. The calculations are computed by relating drug dosage to basal metabolic rate (BMR), also expressed as minimum energy cost (MEC).

Not all drugs are metabolized according to metabolic weight, however. Etorphine is an example. The total dose necessary to immobilize an adult elephant may be as little as 4 mg; it may require 12 mg to immobilize an eland antelope. Without pharmacokinetic and efficacy studies it is difficult to predict which drug dosages should be allometrically scaled. Dosages for five of six antimicrobials predicted by metabolic scaling did not correlate with pharmacokinetic data. Metabolic scaling predicted lower doses and longer treatment intervals for amikacin, trimethoprim sulfa, oxytetracycline, and ceftiofur, and a higher dose for procaine G penicillin than was determined by pharmacokinetic evaluation.[14,39,44]

Certainly factors other than body size influence drug clearance.[47] At the same core body temperature, the horse has a greater MEC/kg body weight than the elephant.

The horse has a more rapid mean circulation time, higher density of capillaries per unit of a given tissue, more respiratory gas exchange surface, and higher intracellular densities of mitochondria and cytochrome-C per unit of body size than the elephant. All these may have a bearing on drug dosage. Further, allometrically determined dosage regimens may be accurate only for drugs that are cleared unaltered by the liver or kidney.[53] Not all veterinarians are proponents of metabolic scaling and many choose to rely on equine doses when elephant-specific scientific data is lacking.

### Extra-Label Drug Use and Compounding

The use of drugs in any manner that is not in accordance with the label constitutes extra-label use. Human compounding (formulating drugs from bulk pharmaceutical ingredients) is one form of extra-label drug use; using a drug for a species other than one it is approved for is another. Virtually all elephant drug use is extra-label. In the U.S., extra-label drug use is authorized under an amendment to the Animal Medicinal Drug Use Clarification Act (AMDUCA) of 1994. The Food and Drug Administration Center maintains regulatory authority for Veterinary Medicine (FDA-CVM). Extra-label drug use regulations are primarily intended to prevent unwanted drug residues from entering the human food chain. Restrictions for nonfood animals are less stringent; however, veterinarians that treat elephants should be familiar with AMDUCA (http://www.fda.gov/cvm/amducatoc.htm).

Compounding from approved drugs must be implemented in compliance with AMDUCA and the FDA Compliance Policy Guide 608.400 ("Compounding of Drugs for Use in Animals"). At the time of this writing, this document is under revision and the current version should be consulted (see www.avma.org). According to the current AVMA position statement (http://www.avma.org/compounding/compounding.asp), compounding should be restricted to drugs that have demonstrated safety and efficacy in the compounded form for the target species or those cases for which no other method or route of drug delivery is possible. Elephants generally fall into this latter category, and compounded drugs have been used to treat numerous conditions in elephants, most notably tuberculosis.[33] There are several veterinary pharmacies in the U.S. that have experience compounding medications for elephants. These are listed in Table 15.1.

## METHODS OF ADMINISTRATION

Regardless of the medication technique selected, the safety of all staff involved must be a primary consideration.[23]

### Oral

The most simple oral delivery method is to mix the medication with food; however, spillage or separation are inevitable, and determining the amount of drug consumed may be impossible if medicated food is offered free choice. It is not practical to administer drugs in the drinking water for the same reason and also because elephants spray their water. Elephants have a well-developed sense of taste and are notorious for their refusal of oral medications. See a discussion of taste in Chapter 32.

Medicated food may be fed directly to some elephants. A great deal of experimentation may be necessary to identify suitable food disguises, and these may vary considerably between individuals. Chocolate and mint flavors may mask bitter tastes and most elephants seem fond of sweets. Wedges can be cut in items such as apples, pineapples, bananas, and papayas to create a space for tablets or liquids. Bread, donuts, jello, popsicles, cola drinks, peanut butter, and sweetened rice balls are among the countless items that have been used successfully (and unsuccessfully). If medication is cooked or frozen with food, it is advisable to determine that such alterations do not change the chemical properties or the stability of the drug. Consult pharmacology texts for information on storage, stability, and compatibility.

**Table 15.1.** Selected Veterinary Compounding Pharmacies

Abbott's Compounding Pharmacy Inc.
2320 Woolsey Street, Suite 105
Berkely, CA 94705-1974
Contact: John Garcia
Tel: 510-548-8777

Bruderer Drugs
26611 N. Dixie Highway
Suite #119
Perrysburgh, OH 43551
Tel: 419-873-2800

Carr Drugs
3801 General DeGaulle
Algiers, LA
Contact: Randy Carr
Tel: 504-367-5724; Fax: 504-367-9475
Email: docrph@aol.com

Congaree Veterinary Pharmacy
1309-B State Street
Cayce, SC 29033
Contact: Terry Fiffick
Tel: 877-939-1335 (toll free); Fax (803)-939-0073
Email: congaree@ix.netcom.com

People's Pharmacy
785 E. Brookhaven Circle
Memphis, TN 38117
Tel: 901-682-2273

Premier Pharmacy
8269 Commercial Way
Weekiwachee, FL 34613
Contact: Vern Allen
Tel: 800-752-7139

Spectrum Chemical Mfg. Corp.
14422 S. San Pedro Street
Gardena, CA 90248
Tel: 310-516-8000 or 800-342-6615
For bulk chemicals, must order 25 kg minimum

Village Compounding Pharmacy
898 Tanager Street
Incline Village, Nevada 89451
Contact: Jonathan Paul Carlier
Pharmacy Education Director
Tel: 800-778-8684; Fax: 775-831-2228
Email: villagepharmacyguy@sbcglobal.net

***Figure 15.1.*** *Most elephants may be readily conditioned to accept a bite block for the direct administration of oral medications.*

It is preferable to deliver oral medications directly. The occasional elephant will open its mouth and permit medications to be placed into the oral cavity but this is rare. Even if successful at first try, this may not be repeatable, particularly with ill-tasting drugs. Elephants may be more likely to accept medications from a trusted handler than from the veterinarian. Caution should always be exercised because even gentle elephants may respond quickly to a sudden movement or distasteful food and crush a hand.

Bite blocks may be used if the elephant has been trained to accept the procedure. The center hole is designed to permit the hand or a tube to pass through (see Fig. 15.1). Care must be taken that the force of the tongue does not push the hand to the side between the molars. Liquid medications may be given with a large animal dose syringe and tubing.

The veterinarian may benefit from working with a pharmacy that has experience successfully compounding medications for elephants. In one case, a 5 g glucosamine treat was prepared as a chewy candy (similar to a Tootsie Roll but four times larger) and was readily accepted by an elephant that had declined numerous other ploys (personal communication, Dr. Jackie Gai, California, May 2005).

## Rectal Administration

The rectal mucosa appears to provide an absorptive surface comparable to the upper gastrointestinal tract. Rectal administration is an alternative route when oral acceptance is poor. Most elephants can be conditioned to accept this method. A procedure is presented in Table 15.2. Therapeutic plasma levels have been demonstrated with metronidazole suppositories.[20] Plasma levels achieved with certain antituberculosis drugs have been comparable to those attained by oral administration.[30,31,72]

## Injection

Despite their large size and thick skin, elephants are quite sensitive to injections. Repeated injections on a long-term basis may not be tolerated. The training and personality of the elephant, the restraint facilities available, and the confidence and skill of the handler determine the ease of administering medications by injec-

**Table 15.2.** Procedure for Rectal Administration

**Equipment**

Disposable large animal OB sleeve
Lubricant
Large animal dose syringe (400 ml)
Flexible polyethylene tubing (used to worm horses); approximately 75 cm (2.5 ft) long
Medication
Warm water

**Procedure**

1. Place the medication (tablets and/or powder) in a container with a lid and dissolve in approximately 60 ml warm water.
2. Apply ample lubrication to the OB sleeve and remove all feces from the rectum.
3. Immediately before administration of the medicated solution, add an additional 40–60 ml warm water, mix thoroughly, and then draw into the dose syringe. The solution should be body temperature (97.5–99°F; 36–37°C), not hot.
4. Attach the tube to the end of the dose syringe. Holding the end of the tube in one hand, insert arm into the rectum. Pass the tubing as far up the rectum as possible and then withdraw arm, keeping the tube in place.
5. Depress the plunger, injecting the medicated solution into the rectum. With the tube still in the rectum, unscrew the syringe, fill with air, and then reattach and inject the air to flush any remaining solution from the tube.
6. Remove the tubing from the rectum by pulling the syringe in an upward direction to empty any residual medication in the tube as it is withdrawn from the rectum.

[a]Developed by Heidi Riddle, Riddle's Elephant and Wildlife Sanctuary, Greenbrier, Arkansas, USA, www.elephantsanctuary.org.

tion. The choice of drugs is limited and the calculated volume of some commercial drugs may be so large as to preclude practical use.

Using an elephant restraint device (ERD) when giving injections may enhance operator safety. If an ERD is not available, the next best option may be to position the elephant in lateral recumbency. Another alternative is for the handler to ask the elephant to hold up one leg to slow reaction time. Although it may be safer to give an injection when the elephant is lying down, in the author's experience, assurance that the medication is being deposited in the muscle and not the SQ tissue is more likely when the injection is given in a weight-bearing leg. In field situations, mahouts are often skilled at rope restraint and elephants may be tethered to trees to restrict movement. A figure-8 rope applied to the hind legs can both restrict movement and prevent kicking.

Pole syringes (jabsticks) may be used for remote injection. Models with spring loading are more effective as medication is injected rapidly (<1 second). The addition of an extension pole on some models can further increase the elephant-to-operator distance. A disadvantage is that the syringe volume is generally 10 cc or less, requiring repeated injections. In dangerous elephants where other options are not available, remote projectors and darts may need to be employed. This equipment is discussed in Chapter 9.

***Figure 15.2.*** *Elephant skin may exceed 2.5 cm (1 inch) in thickness in some areas, such as hip and upper thigh (illustrated here in an Asian elephant). It is important to use needles of the appropriate length when administering intramuscular injections (photo by Hank Hammatt).*

**Intramuscular.** Elephants form abscesses readily so it is important to thoroughly clean the skin prior to giving an injection. Injections should be given into large muscle masses. Commonly used sites include the lateral foreleg (triceps muscles), the hindleg (rectus femoris), and the hip. The skin on the hip and upper hindleg is thicker (≥2.5 cm) than the skin on the foreleg (see Fig. 15.2).

Depress the plunger of the syringe slowly to avoid pain and trauma to the underlying tissue. Swelling may result from injections given too fast. It is helpful to insert the needle first after gently slapping the skin a few times, and then attach the syringe. To insure injection into the muscle, needles of sufficient length must be used. Needles 1.5 inches (3.75 cm) long may be adequate for foreleg injections, but 2.0–3.0 inch (5.08–7.62 cm) may be needed to reach muscle in the hindlegs of adult elephants. Standard hypodermic needles are generally not supplied in lengths greater than 1.5 inches (3.75 cm); spinal needles may be used to achieve the appropriate injection depth; 18 or 20 gauge needles are adequate for most injections. Larger gauge (14 or 16) needles should be reserved for thick drugs because needles this large may be more difficult to place and cause more discomfort.

Recommended maximum volumes per injection site are ≤25 ml,[43] and some clinicians recommend that only 10–20 ml be given.[57] If larger volumes are given, inflammation may result. Dimethyl sulfoxide (DMSO) applied to injection sites may reduce swelling in these situations.[57]

**Subcutaneous.** The subcutaneous route is not recommended unless the drug is specifically labeled to be given by this route and not intramuscularly. Even then,

absorption from subcutaneous tissues in elephants has not been studied and is questionable. Use of short needles may inadvertently result in subcutaneous injection.

**Intravenous.** Intravenous medications may be administered using the auricular, cephalic, or saphenous veins. Some intravenous drugs may be irritating when injected perivascularly. Care should be taken to insure that the needle is securely in the vein, particularly when using the auricular veins. Intravenous injections may be administered with a syringe and needle, but winged infusion sets (butterfly catheters) are recommended for larger volumes. The tubing permits the operator to follow any movements of the elephant without jeopardizing the position of the needle in the vein. Intravenous catheters may be placed for repeated injections, but the elephant must be under constant supervision to prevent their removal.

## Anaphylaxis

Anaphylaxis is a multisystemic, life-threatening, allergic reaction that requires emergency treatment. Anaphylactic reactions have not been reported in elephants and are likely rare, but the clinician should be prepared for this possibility when administering injectable medications or vaccinations. Signs in domestic species include anxiety, muscle tremors, convulsions, dyspnea, tachycardia, profuse salivation, diarrhea, bloat, sudden collapse, and death. Signs generally occur within 2–20 minutes of administration of the inciting agent. Treatment for anaphylaxis must be given promptly. There is no specific treatment for elephants. Slow IV administration of dilute epinephrine (1:10,000; 0.1mg/ml) is advised for domestic large animals at a dosage of 0.01 mg/kg (equivalent to about 5 cc for a horse).[68] Epinephrine may be given IM or SQ if venous access cannot be achieved or in less severe reactions. In this case, a horse would be given 5–10 ml of a 1:1000 (1 mg/ml) concentration.[68] The dose should be adjusted accordingly, depending on the size of the elephant. Epinephrine may need to be repeated at 15-minute intervals. Additionally, corticosteroids, antihistamines, and fluids may be beneficial. Methylprednisolone sodium succinate (1–2 mg/kg IV or IM) or dexamethasone (0.25–1mg/kg IV or IM) and diphenhydramine (0.25–1 mg/kg IV or IM) are recommended for domestic large animals.[68]

Regional digital intravenous perfusion (RDIP). In RDIP, local anesthetics and antibiotics are administered IV under pressure by injection into a peripheral leg vein that has been distended by a tourniquet. The technique was developed in domestic species as a treatment for osteomyelitis and septic arthritis. The tourniquet is left in place for 20–30 minutes. A pneumatic tourniquet (CDA Products, Potter Valley, California, USA, 707-743-1300) was used to administer palliative therapy to a 45-year-old female African elephant with advanced phalangeal osteomyelitis. Multiple treatments over a 7-month period halted the progression, and the procedure was well tolerated.[62] In another case, a sole abscess resolved successfully following two RDIP treatments at 15-day intervals in conjunction with oral antibiotics and antiinflammatory drugs.[42] In this case a rope tourniquet was used and seemed to be adequate.

## Aerosol

Aerosol or inhalation therapy delivers drugs directly into the respiratory system. Local deposition facilitates lower doses and reduced systemic toxicity. Delivery methods include nebulization, dry powder inhalers, and insufflation. Aerosol therapy is used in horses to treat various respiratory disorders.[15,16,26] Inhalation is under investigation as a possible method to treat human tuberculosis (TB). In a recent study, the aerosol administration of nanoparticle encapsulated rifampin, isoniazid, and pyrazinaminde to guinea pigs maintained therapeutic plasma for 6–8 days in plasma and 9–11 days in the lungs.[45] There are no reports of aerosol therapy in elephants, but research is warranted because this modality could potentially reduce drug dose and cost for the treatment of TB in elephants.

## Topical Treatment

Topical treatment is commonly used for superficial wounds. The area should be thoroughly cleaned prior to application of the chosen agent. Elephants will often investigate and remove topical medications, so maintaining adequate contact time may be difficult. Simply keeping wounds clean may be sufficient and should be done frequently. Topical agents are discussed below.

**Fomentation (compresses).** *Fomentation* is the application of moist warm compresses. It is used to reduce pain and swelling or to draw infection to the surface. In Thailand, herbal fomentation for musculoskeletal pain is applied using a towel on the end of a stick (see Fig. 15.3). A portable (dry) heating pad may be fashioned by filling a fine mesh bag with uncooked rice and heating it in the microwave for 2–3 minutes. The rice retains heat for some time and may be reused. Always exercise care to avoid burns when applying warm compresses. If the compress is too hot for human comfort it will be too hot for the elephant. Cold compresses may also be used—especially to reduce acute swelling.

## Wound Lavage (Irrigation, Flushing)

Irrigation is a widely used wound treatment technique. Controlled wound irrigation is less traumatic to tissue, less painful, and more effective in removing foreign material and reducing the bacterial load than swabbing. A variety of lavage solutions are used including sterile saline, dilute povidone iodine, dilute chlorhexidine, and others. Devices to administer wound irrigation in-

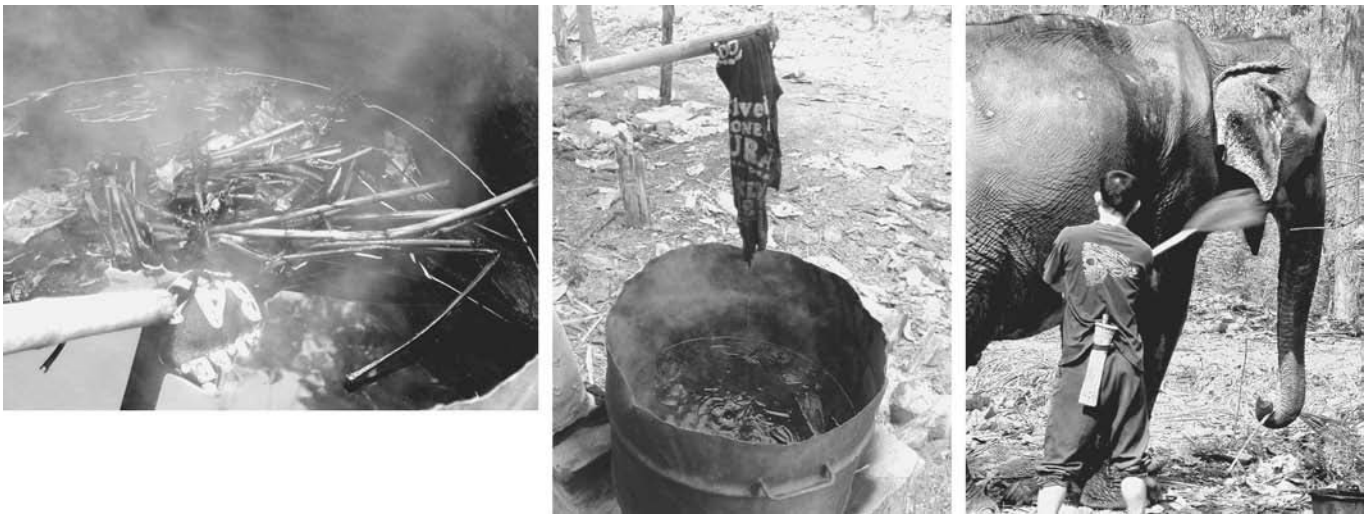

***Figure 15.3.*** *Fomentation (the application of warm compresses) being applied to an elephant in Thailand (photo by Hank Hammatt).*

clude a raised fluid bag with attached tubing permitting gravity flow, bulb syringes (a large basting syringe used in cooking, for example), 60 cc catheter-tipped syringes, and handheld or backpack style garden sprayers. It is important to use copious amounts of the lavage solution—at least 250 ml. Low pressure is ineffective in removing contaminants, and high pressure can damage tissue and force bacteria deeper into the wound. Moderate pressure (9–25 psi) is best.[35]

**Soaking.** Soaking is generally applied to foot injuries and requires some training of the elephant to accept the procedure. After this is accomplished, cooperation is generally good because the procedure is usually pleasurable. A variety of agents may be used. These are discussed with foot disorders in Chapter 20 (see Fig. 15.4).

## Ophthalmic Treatment

Most elephants resent having the area around their eyes manipulated, and it is difficult if not impossible to force their eyelids open. Long eyelashes on some elephants further complicate treatment as does the frequency with which most preparations must be administered. Ophthalmic preparations are available as drops or ointments. The choice of which form to use is best determined by the personality of the elephant and the skill of the handler to apply the product.

Using a small (cosmetic or purse-size) spray bottle has been suggested as a more effective method to administer eye drops.[10] Commercial eye drop products may be transferred into the spray bottle, or solutions may be formulated (by mixing gentamicin with sterile saline, for example, in dilutions equivalent to the commercial product). The elephant handler or mahout may carry the spray bottle and apply the medication opportunistically throughout the day.

## Bandaging

Wounds on lower limbs may be bandaged, but elephants rarely leave bandages in place for long. A number of bandaging techniques, primarily for feet, have been described. Refer to Chapter 20.

***Figure 15.4.*** *Soaking is commonly used in the treatment of foot disorders. A list of solutions is provided in Chapter 20 (photo by Hank Hammatt).*

## ANESTHETICS AND SEDATIVES

Anesthetics and sedatives are discussed in Chapter 9.

## ANTHELMINTICS

Anthelmintics are discussed in Chapter 12.

## ANTIBIOTICS

### Criteria for Selection and Administration

Antibiotics should not be used indiscriminately. Although many organisms demonstrate predictable susceptibility patterns, there are enough variations that selection should be based on results of in vitro sensitivity testing whenever possible. Prophylactic use of antibiotics is both controversial and unproven for domestic large animals.[5] It is not common practice for elephants, but it may be indicated for contaminated wounds or surgical procedures.

Antibiotics may be given orally or by injection. Topical administration may be useful for wounds. Local administration may be considered for bone or joint infections. Antibiotic-impregnated polymethylmethacrylate has been used for musculoskeletal infections in horses, but it has not been described for elephants.[36] In horses, synovial fluid levels of gentamicin (150 mg) or amikacin (250–500 mg) injected intraarticularly may stay above the MIC for over 24 h.[36] Antibiotics may also be delivered under pressure (see the section on regional digital intravenous perfusion (RDIP) above). Reference dosages for antibiotics and antiviral drugs are presented in Table 15.3.

**Table 15.3.** Antimicrobial Reference Dosages for Elephants (See Disclaimer Below)

| Drug | Dosage Guideline | Route | Comments | Reference |
|---|---|---|---|---|
| **Aminoglycosides** | | | | |
| amikacin | EL: 6–8 mg/kg | IM q 24 h | | Lodwick 1994 |
| | EQ: 4.4–6.6 mg/kg | IV or IM q 8–12 h | | Van Erck 2005 |
| | EQ: 21 mg/kg | IV or IM q 24 h | Monitor serum levels. | Plumb 2002 |
| gentamicin | EL: 4.4 mg/kg | IV or IM q 24 h | | Schmidt personal communication in Olsen 1999 |
| | EQ: 6.6 mg/kg | IV or IM q 24 h | Monitor serum levels. | Plumb 2002 |
| kanamycin | EQ: 7.5 mg/kg | IV or IM q 8 h | | Van Erck 2005 |
| **Cephalosporins** | | | | |
| ceftiofur | EL: 2.2 mg/kg | IM q 12–24 h | | Dumonceaux 2003 |
| | EQ: 2.2–4.4 mg/kg | IM q 12–24 h | | Plumb 2002 |
| **Fluorquinolones** | | | | |
| enrofloxacin | EQ: 2.5–5.0 mg/kg | PO or IV q 12 h | | Van Erck 2005 |
| marbofloxacin | EQ: 2 mg/kg | IV, SQ, or PO q 24 h | | Van Erck 2005 |
| **Macrolides** | | | | |
| chloramphenicol | EQ:55 mg/kg | PO q 6 h | | Plumb 2002 |
| chloramphenicol sodium succinate | EQ: 45–60 mg/kg | SQ, IM, or IV q 6–8 h | | Plumb 2002 |
| erythromycin | EQ: 25 mg/kg | PO q 24 h | | Van Erck 2005 |
| metronidazole | EL: 15 mg/kg | per rectum q 24 h | | Gulland 1987 |
| **Penicillins** | | | | |
| amoxicillin | EL: 11 mg/kg | IM q 24 h | | Schmidt 1978 |
| ampicillin | EL: 8 mg/kg | PO q 8–12h | | Rosin 1993 |
| procaine pen G + benzathine | EL: 4,545 IU/kg | IM q 24–96 h | See text for details. | Schmidt 1978 |
| | EL: 2,273 IU/kg | IM q 48 h | See text for details. | Schmidt 1978 |
| **Sulfonamides** | | | | |
| trimethoprim8/-sulfamethoxazole | EL: 22 mg/kg | PO q 12 h | Dose may differ for Asian elephants. | Page 1991 |
| | EQ: 15–30 mg/kg | PO q 12 h | | Plumb 2002 |
| **Tetracyclines** | | | | |
| doxycycline | EQ: 10 mg/kg | PO q 12 h | Do not give IV. | Van Erck 2005 |
| oxytetracycline | EL: 20 mg/kg | IM q 48–72 h | Sensitivity testing is recommended; see text. | Limpoka 1987, Bush 2000 |
| | EQ: 5–10 mg/kg | IV q12h | | Plumb 2002 |
| **Antiviral** | | | | |
| famciclovir | EL: 8–15 mg/kg | PO or per rectum q 8h | | Schmitt 2000; Isaza 2003 |

EL: dosage based on elephant data.

EQ: equine dosage.

**Disclaimer:** This information has been compiled from the scientific literature and is presented as a guideline only. Most elephant dosages are based on studies with limited sample sizes or are anecdotal. See text for further information. Dosages listed may not be appropriate for all elephant species, and specified equine doses may or may not be effective in elephants. The attending veterinarian assumes full responsibility for the use of this information.

## Bacterial Susceptibility Testing

Several methods are used to determine bacterial susceptibility. The agar-disk-diffusion test (ADDT) in which antibiotic-impregnated disks impede bacterial growth on inoculated agar plates is largely qualitative. The minimum inhibitory concentration (MIC) is more precise and is determined by incubating serial antibiotic dilutions in broth with a known number of organisms to determine the lowest concentration of antibiotic that inhibits visible bacterial growth. The minimum bactericidal concentration (MBC) is the lowest concentration of an antibiotic that is lethal and is determined by subculturing an aliquot from the tube containing no visible growth. These latter two techniques are more precise, and coupled with therapeutic drug monitoring may be used as a guide to therapy.

## Postantibiotic Effect

Bacteria may succumb to the effects of antibiotics even when serum concentrations fall below the MIC, an effect demonstrated (in vitro) by aminoglycosides, cephalosporins, penicillins, macrolides, and flouroquinolones.[36] High peak serum concentrations may reduce the bacterial adaptive resistance that may occur with continuous antibiotic contact.[51]

## Therapeutic Drug Monitoring (TDM)

Therapeutic drug monitoring (TDM) is a method in which the serum concentration of a drug from an individual animal is sequentially measured to determine dosage adjustments needed to maintain concentrations within optimal therapeutic ranges. TDM is used in humans with disorders that may alter drug metabolism at ordinary doses (e.g., immunodeficiency, serious organ dysfunction) or to monitor potentially toxic drugs (e.g., aminoglycosides). TDM may be used to guide therapy in elephants where published information is lacking and should especially be considered for serious infections. Pharmacology texts or veterinary or human laboratories should be consulted for optimal sampling times for specific drugs. Serum concentrations may then be compared to ranges known to be effective in other species (horses or humans). In general, serum concentrations should be several times higher than the MIC. The MIC may vary for different target organisms and may also change over time. TDM is required for elephants under treatment for tuberculosis in the U.S.[65]

## Antibiotic Groups

The following is a brief synopsis of antibiotics potentially useful for elephants. It is by no means comprehensive, and veterinary formularies should be consulted for additional details. Olsen[43] has also reviewed antibiotic therapy in elephants.

**Aminoglycosides.** Aminoglycosides are potent, bactericidal antibiotics. Streptomycin and kanamycin are included in this group; however, gentamicin and amikacin are used most frequently. Administration is parenteral. Oral absorption is poor, although an oral gentamicin formulation is available for swine and turkeys. A parenteral dosing frequency of q 24 h achieves higher levels (and greater bacterial kill) in most mammals and may also minimize toxicity. Amikacin is a second-line agent for TB treatment. The intrauterine solution may be of use for local treatment of abscesses in elephants. Due to the potential for toxicity, serum drug levels should be monitored if possible when injectable formulations are used.

**Formulations:** Injectable, oral, intrauterine solution.

**Spectrum: Susceptible:** Gram-negative organisms such as *E. coli* (most), *Enterobacter, Klebsiella, Proteus, Pseudomonas, Salmonella, Serratia, Shigella, Mycoplasma,* and *Mycobacteria* are generally susceptible. **Resistant:** Most anaerobes. *Klebsiella*, *E. coli,* and *Pseudomas aeruginosa* may be resistant to gentamicin.

**Distribution:** Bone; heart; lung; ascitic, pleural, pericardial, peritoneal, synovial, and abscess fluids; some penetration into bronchial secretions; crosses the placenta; variable CSF levels.

**Potential adverse effects**: Ototoxicity, nephrotoxicity (↑ BUN and creatinine), neuromuscular blockade, facial edema, pain or inflammation at injection site. Ototoxicity may be of concern for elephants that must respond to voice commands.

**Elephant data:** In one study, the IV administration of amikacin to two adult female African elephants at a dosage of 8 mg/kg resulted in elimination half-lives of about 4.0 h. Dosages of 3mg/kg and 6 mg/kg administered IM resulted in peak concentrations of 4.8–8.4 µg/ml and 14.2–21.8 µg/ml, respectively. One elephant subsequently given 7 mg/kg IM q 24 h for 21 days developed signs of mild nephrotoxicity (elevated creatinine and casts in the urine) that resolved after cessation of the drug.[29] A dose of 4.4 mg/kg gentamicin IV has been reported to maintain blood levels at 1.7–1.8 µg/ml (Schmidt, personal communication in Olsen[43]).

**Antituberculosis drugs.** Antituberculosis drug research is ongoing and the most recent Guidelines for the Control of Tuberculosis in Elephants (available on the Internet) should be consulted before initiating therapy.[65] The dosages discussed below reflect recommendations at the time of this writing. These antituberculosis drugs are widely distributed in most tissues and fluids (including milk), and cross the placenta. They are available as oral formulations. Isoniazid and rifampin are also available for injection. Serum chemistries should be monitored in elephants receiving anti-TB drugs because of possible hepatotoxic effects. Veterinarians should be familiar with the therapeutic drug monitoring of anti-TB drugs.[48]

Ethambutol (ETH). ETH is a synthetic drug specifically designed for TB treatment. It inhibits bacterial cell wall

synthesis and is bacteriostatic under clinical conditions.[49] ETH has an MIC of 1–5 µg/ml against *M.tb*.[1]

**Spectrum: Susceptible:** Active only against mycobacteria (*M. tb, M. bovis, M. avium, M. kansasii, M. intracellulare).*

**Potential adverse effects:** Optic neuritis may occur in humans.

**Elephant data:** TDM is required to achieve a target serum level of 2–6 µg/ml at 2h. A starting dosage of 30 mg/kg given orally is suggested. Although levels of ETH may be achieved with rectal administration, in preliminary studies (using bulk drug) it appears to be irritating and is rapidly expelled.[31]

Isoniazid (INH). INH is a bacteriocidal synthetic drug. It inhibits bacterial cell wall synthesis. It has an MIC of 0.01–0.25 µg/ml against *M. tuberculosis*.[49]

**Spectrum: Susceptible:** *M. tuberculosis, M. bovis, M. kansasii*, *M. xenopi.* **Resistant:** Most nontuberculous mycobacteria.

**Potential adverse effects**: Anorexia, lethargy, pica, subclinical hepatitis, and anemia have been observed in elephants.[33,65]

**Elephant data:** TDM is required to achieve a target serum level of 3–5 µg/ml at 2h. A starting dosage of 5 mg/kg given orally or by rectum is suggested. Based on pk studies, a dosage of 4 mg/kg may be adequate when INH supplied as powder is administered immediately after suspension.[30]

Pyrazinamide (PZA). PZA is a synthetic antibiotic derived from nicotinic acid. Its mechanism of action is unknown.

**Spectrum: Susceptible:** *M. tb* and *M. africanum.* **Resistant:** *M. bovis* and other mycobacteria.

**Potential adverse effects**: Anorexia, hepatotoxicity; severe epiphora has been noted when PZA was used in combination with enrofloxacin (personal communication, Dr. Genevieve Dumonceaux, Tampa, Florida, May 2005).

**Elephant data:** A starting dose of 30 mg/kg PO or rectally is suggested with a goal of achieving a target serum level of 20–60 µg/ml at 2 h. Pharmacokinetic studies indicate a higher $C_{max}$ and less variability between animals with oral versus rectal dosing.[72]

Rifampin (RIF). RIF is a semisynthetic derivative of rifamycin that interferes with protein synthesis. It is bacteriocidal.

**Spectrum: Susceptible:** *M. tb, M. bovis, M. kansasii*; certain gram-positive and gram-negative bacteria; poxvirus (in high doses).

**Potential adverse effects**: Hepatotoxicity.

**Elephant data:** A starting dose of 10 mg/kg PO is recommended with a goal of achieving a target serum level of 8–24 µg/ml at 2h. Rectal absorption is poor so RIF should be given orally.[33] Rifampin will cause urine, tears, and saliva to turn orange. This has no ill effects.

**Cephalosporins.** Cephalosporins are semisynthetic ß-lactams, similar to the penicillins. They inhibit cell wall synthesis and are usually bacteriocidal. Intramammary preparations may be useful for instillation in wounds in elephants.

**Formulations:** Most are available only as human formulations. Frequent administration (doses specified for horses are often TID or QID) limits practical usefulness for elephants. One exception is ceftiofur, available as a veterinary product for use in cattle and horses.

**Spectrum**: 1st generation. **Susceptible:** Most gram-positive and anaerobic organisms (except *Bacteroides fragilis*); variable gram-negative activity. **Resistant:** Group D *Streptococcus*, methicillin-resistant *Staphylococcus*, indole+ *Proteus, Pseudomonas, Enterobacter, Serratia, Citrobacter;* variable or poor activity against most gram-negative bacteria. 2nd generation: Broader gram-negative and reduced gram-positive activity. 3rd generation: Expanded gram-negative spectrum; gram-positive activity of 1st and 2nd generations retained.

**Distribution:** Widely distributed; bone, pleural, pericardia, synovial fluid, urine, bile; cefotaxime will penetrate CSF; certain others will if meninges inflamed; crosses placenta; enters milk.

**Potential adverse effects:** Hypersensitivity (rash, fever, anaphylaxis), diarrhea.

**Elephant data:** Asian elephants given ceftiofur IV or IM at 1.1 mg/kg showed lower than expected plasma levels, which suggested BID or TID dosing. Increasing the dose to 2.2 mg/kg was recommended, although with concern for the larger volume.[14] In the author's experience a dose of 2.2 mg/kg is achievable in a reasonable volume for an adult elephant by reconstituting the powder formulation (which is readily soluble) in a lesser volume than indicated on the label.

**Fluoroquinolones.** Bacteriocidal (concentration dependent). They are thought to inhibit bacterial DNA synthesis. They show a strong postantibiotic effect and are effective at concentrations 1–4 times the MIC;[36] well absorbed orally in most species.

**Formulations:** Injectable, oral. Ciprofloxacin, a human formulation, is poorly absorbed in horses.

**Spectrum**: **Susceptible:** Most gram-negative bacteria—*Pseudomonas*, *Klebsiella, E. coli, Enterobacter, Campylobacter, Salmonella, Shigella, Proteus, Serratia, Staphylococcus, Mycoplasma, Rickettsia, Mycobacterium*. **Resistant:** *Streptococcus, anaerobes. Pseudomonas, Klebsiella, Acinetobacter,* and enterococci may mutate and become resistant.

**Distribution:** Kidney, liver, bile, skin, bone, CSF, urine, female genital tract.

**Potential adverse effects:** Cartilage abnormalities in young animals and GI distress have been reported

in other species. A 48-year-old Asian cow treated with 2.5 mg/kg enrofloxacin PO for a suspected urinary tract infection elephant exhibited signs of colic after one treatment. No signs of colic were noted before or after this one event (personal communication, Dr. Jackie Gai, California, April 2005). Possible interaction with PZA (see above).

**Elephant data:** There are no published studies. Dosages of 1.5–2.8 mg/kg PO q 24 h and 1.07–1.25 q 12 h have been used anecdotally.[43]

**Macrolides and lincosamides.** These are bacteriostatic antibiotics that disrupt bacterial protein synthesis. This group includes erythromycin, clindamycin, lincomycin, tylosin, and others. Chloramphenicol and metronidazole are sometimes included in this group.

**Formulations:** Injectable, oral, intramammary.

**Spectrum: Susceptible:** Gram-positive cocci (*Staphylococcus, Streptococcus*); gram-positive bacilli (*Corynebacterium, Clostridium*), some gram-negative bacilli, some *Actinomyces* and *Mycoplasma.* **Resistant:** *Pseudomonas, E. coli, Klebsiella*

**Distribution:** Widely distributed, crosses placenta, enters milk, CSF levels poor.

**Potential adverse effects:** Uncommon; local reactions, diarrhea, anorexia.

**Elephant data:** There is no published pk data. Severe gastrointestinal pain has been reported following oral administration of erythromycin (Schmidt, personal communication in Olsen[43]). Tylosin has been used at an anecdotal dosage of 12 mg/kg/day IM for 5 days to treat acute mycoplasma infections.[57]

Chloramphenicol. Chloramphenicol is usually bacteriostatic, but it may be bacteriocidal at high concentrations. It interferes with bacterial protein synthesis. Aplastic anemia in humans has been associated with chloramphenicol, so use caution when handling.

**Formulations:** Injectable, oral.

**Spectrum**: **Susceptible:** many gram-positive and gram-negative bacteria, including *Salmonella, Staphylococcus, Streptococcus, Shigella*; anaerobes, including *Clostridium, Bacteroides, Fusobacterium; Nocardia, Mycoplasma, Rickettsia.*

**Distribution:** CNS, liver, kidney, pleural, peritoneal and synovial fluid, aqueous humor, milk, crosses placenta.

**Potential adverse effects**: Bone marrow suppression with long-term treatment, anorexia, diarrhea, IM injections painful in horses.

**Elephant data:** No published information.

Metronidazole. Metronidazole is a nitroiimidazole antibacterial and antiprotozoal drug. It is bacteriocidal.

**Formulations:** Oral, injectable.

**Spectrum**: **Susceptible:** Anaerobes, including *Clostridium*, *Bacteroides, Fusobacterium.* Resistant: *Actinomyces.*

**Distribution:** Widely distributed to most tissues and fluids including bone, abscesses, CNS.

**Potential adverse effects**: CNS depression.

**Elephant data:** Metronidazole (15 mg/kg q 24 h) given as a rectal suppository to a single elephant achieved plasma concentrations of 4.4–7.7 µg/ml during 24 h after administration, which exceeded the MIC for most anaerobic human pathogens (0.3–3.0 µg/ml).[20]

**Penicillins.** Penicillins are ß-lactam antibiotics that inhibit bacterial cell wall formation. They are bacteriocidal. Several classes comprise this group: natural penicillins (G and K), penicillinase-resistant (e.g., cloaxcillin), amiopenicillins (e.g., ampicillin, amoxicillin), extended spectrum (e.g., carbenicillin, ticaricillin), and potentiated (e.g., amoxicillin-clavulanate).

**Formulations:** Oral, injectable, intramammary. There are numerous penicillin formulations, and the nomenclature is confusing. The procaine and benzathine formulations are depot forms that are hydrolyzed to penicillin G sodium in vivo. Concentration is expressed in units: 1 mg of liquid penicillin G potassium = 1440–1680 USP units; 1 mg of penicillin G sodium = 1500–1750 USP units (units vary for powder formulations). Repository formulations are typically dosed at 24–48 h intervals in cattle.

**Spectrum:** Varies with the group.

Natural penicillins. **Susceptible:** Gram-positive and negative aerobic cocci, some aerobic and anaerobic bacilli (e.g., *Clostridium, Fusobacterium*, and *Actinomyces*). **Resistant:** Most gram-negative bacteria, mycobacteria, *Mycoplasma*.

Aminopenicillins. **Susceptible:** Many gram-negative aerobes, including *Klebsiella*, some strains of *E. coli*, some anaerobes (*Clostridium*). **Resistant:** *Pseudomona aeruginosa,* Serratia, indole + *Proteus, Enterobacter, Citrobacter, Acinetobacter,* mycobacteria, *Mycoplasma*.

**Distribution:** Widely distributed; kidney, heart, liver, skin, bile, bone, ascitic and synovial fluids, crosses placenta, poor CSF penetration.

**Potential adverse effects:** Uncommon; hypersensitivity possible, diarrhea.

**Elephant data:** Procaine penicillin G (150,000 IU/ml) in combination with benzathine penicillin (150,000 IU/ml) was studied in five Asian elephants and resulted in the following dosage recommendations: 1) 4,545 IU/kg IM q 96h or 2,273 IV/kg IM q 48h hours for *Bacillus anthracis, Corynebacterium diphtherae, Streptococci* spp, and nonpenicillinase producing *Staphylococci*; 2) 4,545 IU/kg q 36 h for *Clostridia*; and 3) 4,545 IU/kg q 24 h for *Pasteurella multocida*.[56]

Amoxicillin given at 11 mg/kg to five Asian elephants resulted in maximum serum concentrations of 1.567 mcg/ml. The serum plateau level was reached at 6–8

hours and fell slowly over 12–24 h, suggesting that a dosing interval of q 24 would be adequate.[56]

A single oral dose of ampicillin (8 mg/kg) given to three Asian elephants achieved a mean peak concentration of 0.86 µg/ml at 90 minutes and remained above the MIC of an elephant staphylococcus and streptococcus isolate (MIC = 0.06 µg/ml) for >8h. The authors recommend a dose of 8 mg/kg PO q 8–12 h for susceptible *Staphylococcus, Streptococcus* and *Proteus* but caution that this dose may not be effective against salmonella (MIC = 0.02–2.5 µg /ml) or *E. coli* (MIC = 5.0 µg /ml).[54]

**Sulfonamides.** Sulfonamides and trimethoprim interfere with bacterial thymidine synthesis. In the U.S. the veterinary product combines trimethoprim and sulfadiazine (TMP-SD), and the commonly used human product combines trimethoprim and sulfamethoxazole (TMP-SMZ) These combinations are bacteriocidal and well absorbed orally.

**Formulations:** Injectable, oral.

**Spectrum: Susceptible:** Most gram-positive bacteria and *Nocardia*; anaerobic bacteria, including those isolated from chronic tusk infections;[32] most gram-negative *Enterobacteriaceae, Coccidia*, and *Toxoplasma*. **Resistant:** *Pseudomonas*, most anaerobes.

**Potential adverse effects:** Diarrhea, hypersensitivity.

**Elephant data:** TMP-SMZ was administered IV and orally at a combined dose of 22 mg/kg to three African elephants and one Asian elephant. Pharmacokinetic parameters for African elephants were similar to horses, but values for the Asian elephant differed, suggesting possible species variation. The authors suggest that a dose of 22 mg/kg PO q 12h would be clinically effective, although dosing 4–6 times a day would be necessary to maintain trough levels above the MIC.[44]

**Tetracyclines.** Tetracyclines are bacteriostatic and inhibit protein synthesis. They are absorbed well orally. Doxycycline is semisynthetic and derived from oxytetracycline. It has a longer half-life and higher CNS penetration.

**Formulations:** Oral, injectable.

**Spectrum: Susceptible:** Many gram-positive bacteria, including *Clostridium perfringens* and *C. tetani, Actinomyces, Bacillus anthracis*; gram-negative bacteria, including *Pasteurella, Shigella, Yersinia*. **Resistant:** *E. coli, Klebsiella, Enterobacter, Proteus, Pseudomonas.*

**Distribution:** Widely distributed; lung; heart; kidney; muscle; bile; synovial, ascitic, peritoneal fluid; urine; crosses placenta; enters milk; CSF levels poor.

**Potential adverse effects:** Pain on injection and profound tissue reaction in elephants; diarrhea, transient collapse with rapid IV administration. Fatalities have occurred in horses following IV doxycycline.

**Elephant data:** There have been three pk studies in elephants.

In one study, oxytetracycline in aqueous 2-pyrrolidone was given IV and IM at a dose of 20 mg/kg to six Asian elephants. Following IV administration, an average peak plasma concentration of 6.2 µg /ml was measured at 1h with no detectable drug after 60h. Plasma concentration peaked at 2.87 µg/ml 2 h after IM injection, and concentrations >1 µg/ml were maintained for 48h.[27] In a crossover study, in which doses of 18 mg/kg IM and 8 mg/kg IV were given to 18 African elephant calves under anesthesia, serum oxytetracycline concentrations >0.5 µg/ml were present for 48 h, but only elephants given the high IM dose maintained this level 72 h post injection.[8] This study was conducted in Africa, and the authors point out that therapeutic ranges may vary geographically. At the prescribed dose, using the commercially available product (200 mg/ml), an adult cow weighing 2727 kg (6000 lbs) would require 245 cc. In this author's experience volumes of ~25 ml/site may result in considerable swelling so it is doubtful that this drug is practical for most situations unless given IV.

In the third study, it was concluded that a dosage of 60–80 mg/cm IV or IM should maintain therapeutic levels for 48 h. The weight estimates in this study were based on girth and length measurements but were not related to weight.[7]

## ANTIVIRAL DRUGS

Only one antiviral drug has been reported in elephants. Famciclovir was used successfully to treat three cases of endotheliotrophic herpesvirus infection in two juvenile Asian elephants[58] and a subadult bull.[55] Famciclovir is a pro-drug of penciclovir, a human antiherpes drug. In one juvenile case, treatment was initiated with a loading dose of 12.8 mg/kg po on day 1 followed by 6.4 mg/kg po q 8h. On day 9 the dose was reduced to 4.06 mg/kg q 8 h, based on allometric scaling, and on day 14 it was changed to 6.4 mg/kg po q 12 h for 5 days and then withdrawn. Serum penciclovir levels varied between 97–4365 ng/ml and were within therapeutic human ranges. Oral medication was refused in the second juvenile case and famciclovir (10.6 mg/kg q12 h) was given rectally. The dose was reduced to 6.7 mg/kg on day 3 of treatment and continued for a total of 30 days.[58] Results of a subsequent pharmacokinetic study in young Asian elephants concluded that a dose range of 8–15 mg/kg given PO or rectally should produce concentrations of penciclovir comparable to therapeutic levels in humans.[22]

## ANTIFUNGAL DRUGS

Reports of fungal infections in elephants are infrequent and there are no published studies of antifungal drugs. Fungal infections should be confirmed by culture and topical or systemic treatment initiated following recommendations for horses.

## ANTIINFLAMMATORY DRUGS

Antiinflammatory medications are administered to elephants primarily for the treatment of musculoskeletal disorders, arthritic conditions, and colic. The nonsteroidal antiinflammatory drugs (NSAIDS) are used most commonly, although corticosteroids and dimethylsulfoxide (DMSO) are also in this category. Corticosteroids have not been studied in elephants and the use of DMSO is anecdotal.

### NSAIDS

The NSAIDS have antipyretic, analgesic, and antiinflammatory actions that are dose dependent. Their mechanism of action is similar. NSAIDS inhibit cyclooxygenase and impede the synthesis of prostaglandins. Localized swelling following injection, gastrointestinal ulceration, and renal papillary necrosis are potential side effects. In horses, phenylbutazone has the greatest potential for toxicity, flunixin less, and ketoprofen least.[37] Anecdotal dosages have been reported[39] and there are two published pk studies in elephants.

**Aspirin.** Aspirin (acetylsalicylic acid) is the salicylate ester of acetic acid. It has a short half-life in horses and is used mainly for thromboembolic disorders (e.g., DIC) to decrease platelet aggregation.[37] Elephant platelets are unresponsive to inhibition by aspirin.[18] Aspirin is available in numerous human oral formulations and as a rectal suppository. Veterinary formulations include a molasses-flavored powder and an oral formulation containing 300 mg aspirin and 0.5 mg methylprednisolone per tablet.

**Flunixin meglumine.** Flunixin is widely used in elephants for musculoskeletal pain and colic. It is available as an injectable and for oral use as a paste or granules. It is recommended that treatment not exceed 5 consecutive days in horses. Some studies have associated flunixin with reduced wound healing in horses.[37] Flunixin may be given twice a day for colic and antiendotoxic dosages of 0.25–0.5 mg/kg IV q 6–8 h are used in horses.[51]

**Ibuprofen.** Ibuprofen administered at a dosage of 6 mg/kg PO q 12 h to Asian elephants and 7 mg/kg PO q 12 h to African elephants will achieve serum concentrations of 15–30 µg/ml that are within the therapeutic ranges for humans.[2] Anecdotal reports on the use of ibuprofen have been favorable, and the author is aware of long-term, continuous use (months to years) with no ill effects. In one case, a 38-year-old Asian female elephant with severe arthritis was treated with approximately 6 mg/kg ibuprofen PO BID and 1.25 mg/kg acetaminophen PO BID for 6 months. The elephant seemed more comfortable when this dosage was given compared to lower dosages, and worsening of clinical signs (stiffness, unwillingness to move) occurred when doses were missed. Tablets were ground to a fine powder in a coffee grinder and mixed with fruit punch that the elephant drank voluntarily (personal communication, Dr. Jackie Gai, California, May 2005).

**Ketoprofen.** Ketoprofen administered IV and orally to five Asian elephants was completely absorbed and demonstrated a long terminal half-life.[21] Ketoprofen is comprised of 2 enantiomers with different pharmacokinetic properties although the commercial product is a racemic mixture. The metabolism of the S-enantiomer (thought to be the more active with regard to cyclooxygenase inhibition) was more rapid in elephants than in horses. A dosage of 1–2 mg/kg PO or IV q 24–48 h was suggested. The allometrically determined dosage did not accurately predict the dosage for elephants, emphasizing the importance of pk studies.[21] The long-term safety and efficacy have not been determined. Ketoprofen should not be used concomitantly with aspirin. In one case, ketoprofen was formulated with canola oil, caramel flavoring, and powdered sugar to make a concentrated oral paste that was 6g/35 ml. A topical preparation was also made using pluronic 20% and lecithin organogel to create a gel that could be absorbed through the skin. The gel was applied below and lateral to the tail on an elephant during a long transport when she refused oral medication. Previous indicators of discomfort (weight shifting and holding a foot up) ceased after application (personal communication, Dr. Ann Duncan, Detroit, Michigan, April 2005). Human and veterinary formulations are available.

**Naproxen.** Naproxen appears to have few adverse effects in horses. Its use in elephants has not been reported. Human and veterinary formulations are available.

**Phenylbutazone.** Anecdotal doses of 1–2 mg/kg every 24 hours (route of administration not specified) have been reported based on a survey of 20 U.S. zoo veterinarians.[39] Two cases of segmental gangrene and sloughing of elephants' ears after intravenous injection of phenylbutazone have been reported and more have probably occurred.[34] Phenylbutazone is available in oral tablet, paste, and injectable formulations. Phenylbutazone (2.2 mg/kg IV q 12 h) has been used in horses to inhibit endotoxin effects in cases of endotoxin-mediated ileus.[37]

Reference dosages for antiinflammatory drugs are presented in Table 15.4.

## CHONDROPROTECTANTS AND NUTRACEUTICALS

Both veterinary and over-the-counter products used to treat nonseptic arthritis in horses have gained popular-

**Table 15.4.** Antiinflammatory Reference Dosages for Elephants (See Disclaimer Below)

| Drug | Dosage Guideline | Route and Frequency | Reference |
|---|---|---|---|
| Aspirin | EQ:25 mg/kg | PO q 12 h followed by 10 mg/kg q 24 h | Plumb 2002 |
| Flunixin | EQ:1.1 mg/kg | IV, IM, or PO for up to 5 days; use IV route for colic and repeat as needed | Plumb 2002 |
| Ibuprofen | EL: 6 mg/kg | PO BID (Asian) | Bechert 2003 |
| Ibuprofen | EL: 7 mg/kg | PO BID (African) | Bechert 2003 |
| Ketoprofen | EL: 1–2 mg/kg | PO or IV q 24–48 h | Hunter 2003 |
| Naproxen | EQ: 5 mg/kg IV; 10 mg/kg PO | 5 mg/kg slow IV and then 10 mg/kg PO q 12 h for up to 14 days or 10 mg/kg PO q 12 h for up to 14 days | Plumb 2002 |
| Phenylbutazone | EQ: 4.4–8.8 mg/kg | PO q 24 h | Plumb 2002 |
| Phenylbutazone | EQ: 3–6 mg/kg | IV q 12 h | Plumb 2002 |
| Dexamethasone | EQ: 2.5–5.0 mg/kg | IV or IM | Plumb 2002 |
| Prednisone | EQ: 1 mg/kg | PO q 12 h for 1 week; decrease dose by 25% each week | Van Erck 2005 |

EL: dosage based on elephant data.
EQ: equine dosage.
**Disclaimer:** This information has been compiled from the scientific literature and is presented as a guideline only. Most elephant dosages are based on studies with limited sample sizes or are anecdotal. See text for further information. Dosages listed may not be appropriate for all elephant species, and specified equine doses may or may not be effective in elephants. The attending veterinarian assumes full responsibility for the use of this information.

ity as palliative treatments for degenerative joint disease in elephants. Polysulfated glycosaminoglycan (PSAG) is an analog of mucopolysaccharides found naturally in cartilage that increases the synthesis of proteoglycans and inhibits the proteolytic enzymes that cause their degradation. PSAG has antiinflammatory activity and increases the viscosity of synovial fluid. The only licensed PSAG for veterinary use is Adequan® (Luitpold Pharmaceutical, Inc., Shirley, New York). It is used in horses as an intraarticular or intramuscular injection. The IM equine dose is 500 mg q 4 days for 28 days. Adequan® was used in an African elephant with arthritis of the hip at a dose of 5 g once or twice weekly, with notable clinical improvement and exacerbation of signs during a 4-week period when the drug was not given (personal communication, Dr. Kathyrn Gamble, Chicago Illinois, April 2005).

Hyaluronate sodium, another mucopolysaccharide, is used to treat mild synovitis in the horse; it is given by IV or intraarticular injection. There are no published reports of its use in elephants.

Nutraceuticals (food products with druglike properties) used as chondroprotectants may contain glucosamine (an amino derivative of glucose found in glycoproteins and mucopolysaccharides), chondroitin (a glycosaminoglycan), and/or methylsulfonylmethane (MSM), an organic sulfa made from DMSO**.** An equine product that is a combination of glucosamine, chondroitin, and manganese (Cosequin®, Nutramax Laboratories, Inc, Edgewood, MD 21040, www.nutramaxlabs .com) seems to be well accepted and achieves clinical improvement (personal communication, Dr. Genevieve Dumonceaux, Tampa, Florida, April 2005).

SynoviCre EQ® (DVM Pharmaceuticals, www.dvmpharmaceuticals.com), a combination of glucosamine HCl and creatine monohydrate, resulted in increased ambulation and activity in geriatric Asian elephants with previous signs of joint stiffness. A starting dosage of 8 mg/kg was given for a minimum of 6 weeks and then reduced as signs improved to a maintenance dosage of about 3.5 mg/kg. There were no apparent side effects, even with continuous use over a 4.5-year period (personal communication, Dr. Kelly Helmick, Seattle, Washington, June 2005).

Nutraceuticals appear to have a beneficial effect in elephants with arthritis and further research is warranted.

## FLUID THERAPY

Maintenance fluid requirements have not been determined for elephants. Extrapolating from the horse, adult elephants may need 40–60 ml/kg/24h. Young calves may require 100–120 ml/kg/24h. If dehydration is present, additional fluids will be needed. The volume of replacement fluid (liters) = the body weight (kg) x percent dehydration. With mild dehydration (5–7%), the mucous membranes may be dry, the capillary refill time may be slow, and depression may be evident. Depression will be more pronounced and capillary refill time even slower (2–4 seconds) with moderate dehydration (8–10%). If dehydration exceeds 10%, the elephant will likely be moribund and the capillary refill time may exceed 5 seconds. Elevations in packed cell volume and total protein may help estimate the degree of dehydration and guide therapy.

Lactated Ringers solution is an adequate replacement fluid for most situations. Sequential serum chemistries should be evaluated in all elephants that are ill enough to require fluid therapy, and electrolyte abnormalities should be corrected according to equine guidelines.

Fluid administration may be logistically challenging.

For an average adult elephant weighing 4000 kg, maintenance fluids (40ml/kg) alone would total 160 liters q 24h.[23] Large (12–14 gauge) catheters placed at multiple sites may be necessary to deliver these volumes, and fluid pumps may be helpful. Catheters must be carefully placed (and secured by suturing and/or bandaging) and the elephant monitored closely to prevent removal. Rectal administration may be used as an adjunct to IV therapy.

## TOPICAL AGENTS

Topical agents may be in the form of solutions, ointments, gels, powders, sprays, or pastes. They may be used to reduce bacterial contamination, protect underlying tissue, maintain moisture, promote drying, break down necrotic tissues, deter flies, or enhance healing. The agent chosen may vary with the nature of the wound, how often treatment can be administered, the degree of contamination or infection, and the elephant's level of cooperation. Numerous studies have compared the effects of various agents on neovascularization, epithelialization, and other aspects of wound healing, often with conflicting results.[6,19,25]

Keeping a wound clean is the most important aspect of wound management. Small garden sprayers are useful for this purpose and can be used with saline or antibacterial solutions. If used with povidone iodine, the unit should be flushed after each use because the iodine seems to affect the rubber and may precipitate. Polyethylene tubing or feeding tube catheters may be attached to some units for flushing directly into a wound or abscess cavity.

Some ointments adhere better than others. Zinc oxide ointment has good adhering qualities and encourages granulation tissue and powder-based products (such as Wonder Dust®, Farnam, Phoenix, Arizona) work well for leg wounds. In the author's experience, Preparation H® (Whitehall-Robins Healthcare, Madison NJ) is very effective for superficial wounds to encourage granulation. It can be used alone or mixed 1:1 with any topical antibacterial ointment (such as silver sulfadiazine) for contaminated wounds. Preparation H® contains substances that stimulate oxygen consumption and encourage epithelialization and collagen synthesis.[63] Ointment may be placed in a 60 cc catheter-tipped syringe for instillation into deep wounds. Selected topical agents are listed in Table 15.5.

### Topical Ophthalmic Preparations

There are no specific ophthalmic preparations recommended for elephants. Ointments may be preferred for their longer duration of activity compared to drops because instillation into the eye can be challenging. Alternatively, a small spray bottle can be devised. See administration techniques above.

## VACCINES

There are no vaccines specifically approved for use in elephants. The prevalence of specific diseases in particular geographic areas may determine which vaccines to consider. See Table 7.1 in Chapter 7 for further information.

## EUTHANASIA AGENTS

There are no specific euthanasia agents recommended for elephants. In the U.S. euthanasia should be in accordance with AVMA guidelines, which are updated periodically (see www.avma.org). Due to the elephant's large volume of distribution, agents administered IV may not act as quickly as in other species. For this reason, it is advisable to sedate the elephant heavily with a drug such as xylazine before administering the euthanasia agent. See further discussion in Chapter 33.

## ADVERSE DRUG EFFECTS

Adverse effects have been reported for several drugs that are used in elephants. Anecdotal observations have already been mentioned in the text. Published reports are listed in Table 15.6.

## COMPLEMENTARY OR ALTERNATIVE MEDICINE

Brief mention will be made of a few alternative systems of veterinary care that predate modern (20th century) conventional veterinary medicine. Clinicians are encouraged to maintain an open mind to systems that differ from that in which they were trained. Complementary or alternative medicine (CAM) is gaining attention in the U.S., and some veterinary schools even offer courses. Whereas Western (allopathic) medicine attributes disease to external factors, many other systems share the different view that it is the disruption of the body's natural vital energy that is the underlying basis of disease. Therapeutics are *holistic* (by definition, integrating body, mind, and spirit) and aimed at stimulating the body's innate healing abilities to restore balance rather than at eliminating an intruding agent.

Some traditional therapies may seem irreconcilable with modern scientific thought. For example, bacteria and viruses, commonly acknowledged pathogens in Western medicine, are not recognized in Traditional Chinese Veterinary Medicine (TCVM).[46] The concept of balance, however, is one that even Western medicine is beginning to embrace. Zoo and wildlife veterinarians have long recognized the impact of environment, nutrition, and stress on health, longevity, and reproduction in nondomestic species. In reality, most treatments used on elephants lack scientific proof regardless of the system from which they originate.

**Table 15.5.** Selected Topical Wound Treatment Agents

| Name | Class | Spectrum | Indications | Formulation | Advantages | Disadvantages |
|---|---|---|---|---|---|---|
| Aloe vera | antimicrobial | includes *Pseudomonas* | topical wound application | | enhances epithelialization | antiinflammatory properties may slow wound healing |
| Bacitracin-neomycin-polymixin | antimicrobial | broad spectrum; particularly *Staphylococcus* and *Streptococcus*; *Pseudomonas* is resistant | topical wound application | ointment | encourages epithelialization; no systemic absorption | |
| Cephalosporin | antimicrobial | gram-positive and gram-negative bacteria | wound instillation | intramammary | potential local effect | |
| Chlorhexidine (CH) | antimicrobial | broad spectrum; some g- are resistant (e.g., *Proteus*, *Serratia,* and *Pseudomonas*) | lavage | Use diacetate or digluconate form diluted to 0.05% solution (mix 1 part 2% CH with 40 parts water). | good residual activity (48 h) even in the presence of organic matter; relatively nontoxic | |
| Dakin's solution (Sodium hypochlorite) | antimicrobial | very effective against *Staphylococcus* | lavage | Use as a 0.25% or 0.125% solution. For a 0.25% solution, mix 50 ml bleach (5%)+ 30 ml sodium bicarbonate; qs to 1000 ml. | helps mobilize tissue debris | inactivated by light and deteriorates after a few days so best to mix fresh each time |
| Dimethylsulfoxide (DMSO) | antiinflammatory | NA | topical wound application | gel | may reduce wound swelling or effects of perivascular administration of caustic drugs | drying, epithelial desquamation; absorbed by human skin—wear gloves when applying |
| Gentamicin sulfate | antimicrobial | broad spectrum (effective against *Proteus*, *Pseudomonas,* and *E.coli*) | topical wound application | 0.1% ointment | | may slow healing |
| Honey | antimicrobial | broad spectrum (including *Staphylococcus*, *Streptococcus*, *Salmonella*, *Pseudomonas*) | topical wound application | Use unpasteurized honey—manuka, floral, or lime honey reported to be the most effective varieties. | | effectiveness may vary with honey strain |
| Hydrogen peroxide | mechanical cleanser | more effective against gram-positive than gram-negative bacteria, but antibacterial effect is minimal compared to other agents; will kill some spores | lavage | Commercial product is 3% and is best diluted. | dissolves clots | may injure cells |
| Lactated Ringers solution | mechanical cleanser | NA | reduce bacterial numbers | | physiologic; no tissue damage | no antimicrobial properties; possible tissue edema if fluids retained |

(*continued*)

**Table 15.5.** Selected Topical Wound Treatment Agents

| Name | Class | Spectrum | Indications | Formulation | Advantages | Disadvantages |
|---|---|---|---|---|---|---|
| Live yeast cell derivative (hemorrhoidal ointment Preparation H®; Whitehall-Robins Healthcare, Madison, NJ) | stimulate healing | NA | | Use alone to encourage granulation tissue or mixed 1:1 with a topical antibacterial if infection is present. | encourages oxygen consumption, epithelialization, collagen synthesis and formation of granulation bed | |
| Nitrofurazone | antimicrobial | gram-positive and gram-negative bacteria; *Pseudomonas* resistant | topical wound application | ointment, powder, solution | | |
| Povidone iodine | antimicrobial | broad spectrum; bacteria, mycobacteria, fungi, viruses | lavage; topical wound application | For lavage, dilute to 1% (mix 1 part 10% PI solution with 9 parts sterile water); ointment, cream. avoid detergent formulations (scrubs) | few bacteria are resistant; lower dilutions more effective than concentrated solution | duration of bacterial effect short (4–6 h); inactivated by blood, exudates, and soil; some reports indicate cytotoxicity |
| Penicillin | antimicrobial | gram positive and aerobes and anaerobes | wound instillation | intramammary | potential local effect | limited spectrum |
| Saline | mechanical cleanser | NA | lavage | intramammary | physiologic; no tissue damage | no antimicrobial properties; possible tissue edema if fluids retained |
| Scarlet oil | antiseptic | | topical wound application | solution | stimulates epithelialization | |
| Silver sulfadiazine | antimicrobial | broad spectrum (many gram positive and gram negative bacteria (including *Pseudomonas*); fungi | topical wound application | ointment or cream | good for burns; minimal toxicity; promotes epithelialization; penetrates necrotic tissue | |
| SWAT® (Farnam, Phoenix, AZ) | fly repellent | NA | topical wound application | ointment | nonirritating; may be used near open wounds | |
| Trypsin-Balsam of Peru-Castor Oil (Granulex®) (Pfizer, New York, NY) | enzyme | NA | wound debridement | spray | balsam of Peru increases local circulation; castor oil encourages epithelialization; trypsin digests necrotic tissue | may sting |
| Wonder Dust® (Farnam, Phoenix, AZ) | antiseptic, drying agent, blood coagulant | | topical wound application | powder; contains flowers of sulfur, copper sulfate, hydrated lime, iodoform, and other ingredients | "puffer" dispenser allows application from a short distance; adheres well; good for superficial leg wounds | |
| Zinc oxide | antiseptic | | topical wound application | ointment | | |

References: Swaim 1987, 1990; Burks 1998; Willix 1992; Cooper 1999a, 1999b; Liptak 1997.

**Table 15.6.** Drugs with Reported Side Effects in Elephants*

| Drug | Reported Side Effects |
|---|---|
| Acepromazine maleate | Photosensitization (appearing first as a triangle on the dorsal aspect of the neck) in Asian elephants given acepromazine and xylazine and exposed to direct sunlight during transport (Cheeran 2002). |
| Atropine sulfate | An Asian elephant sedated with azaperone (0.35 mg/kg) IM and atropine (.05 mg/kg) IV became agitated within a minute of the injection of atropine (swaying, kicking, refusing to obey commands). When the behavior did not abate after 30 minutes, azaperone (0.018 mg/kg) was administered IM; the elephant became calm and responsive to commands within 15 minutes. The authors suggest a drug interaction with azaperone, toxicosis due to a dead calf, or species differences as possible causative factors (Gross, 1994). |
| Azaperone | Two abnormal responses were seen in the same cow during azaperone induction. The episodes of confused or hallucinatory behavior were responses to mild stimuli. When maximum effect was attained, no further problems were noted (Schmitt, 1996). |
| Cephalosporins | Potential side effects (renal and hepatic problems) have been noted when a second generation cephalosporin was used IV (specific drug not cited) (Schmidt 1986). |
| Erythromycin | Severe gastrointestinal pain and upset may result after only 1–2 days (Schmidt 1986). |
| Isoniazid | Anorexia, lethargy, pica, yellow-brown urine, elevated liver enzymes (AST, bilirubin, bile acids, LDH, GGT), anemia, decreased white blood cell count.<br>Signs may be dose related; some signs may resolve by a short-term interrupting treatment (a few days to one week) (Mikota 2001).<br>Peripheral nerve damage may be associated with INH in humans but has not been reported in elephants (USDA 2003).<br>Note that most antituberculosis drugs are given concurrently and it may be difficult to associate a specific side effect with a single drug. |
| Ketamine | A single case of photosensitization (similar to that noted with the use of acepromazine) was seen in an Asian elephant sedated with ketamine-xylazine (Cheeran 2002). |
| Tetramisole | Muscle tremors, salivation, bradycardia, and diarrhea followed by constipation were noted in a baby elephant treated with tetramisole; signs resolved following treatment with dextrose, B complex, atropine, and liquid parrafin (Gnanaprakasam 1992). |
| Phenylbutazone | Segmental gangrene and sloughing of ears after intravenous injection (Miller 1977). |
| Pyrazinamide | Anorexia, hepatotoxicity; anemia noted in an elephant receiving PZA and INH (Mikota 2001);<br>severe epiphora when used in combination with enrofloxacin (Dumonceaux 2005).<br>Note that most antituberculosis drugs are given concurrently and it may be difficult to associate a specific side effect with a single drug. |
| Rifampin | Anorexia, lethargy, pica (Mikota 2001).<br>Hepatitis is the major toxicity in humans (USDA 2003). |
| Xylazine | A 27-year-old male Asian elephant with mild bilateral corneal opacities was laid in lateral recumbency; 150 mg xylazine was given slowly IV; and he was then allowed to stand. After 2.5 minutes he tilted his head upward and backward in a tentative gait. At 4–5 minutes he trumpeted loudly and started to shake his head vigorously, followed by complete delirium. After 10 minutes of violent excitement, he gradually became normal but his degree of sedation was minimal. An additional 50 mg of xylazine was given IV and the elephant became profoundly sedated. He was reversed with 60 mg yohimbine IV. The author suggests that the elephant's visual impairment may have caused the reaction (Sarma 1999). |

*For further information see the *Elephant Care International Elephant Formulary* (http://www.elephantcare.org/drugdos.htm).

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> It matters not whether medicine is old or new, so long as it brings about a cure.
> It matters not whether theories are Eastern or Western so long as they prove to be true.
>
> Liu Jian Yi

Veterinarians are encouraged to work closely with elephant caretakers to identify all of the factors involved in the creation of "dis"-ease. Remedying underlying environmental, psychological, or social stressors may be as important to the healing process as the drugs that are chosen to alleviate the clinical signs.

Further scientific investigation into the treatment of elephant diseases is needed. It is critical that we continue to publish scientific articles, but anecdotal information has been and will continue to be of great value. Those of us that care for elephants have a profound responsibility to share what we have learned—both what has been effective and what has not—to improve our care of these endangered species.

## Ayurveda

Sanskrit for "knowledge of life," ayurveda is a 5000-year-old system still widely practiced in India today. According to ayurveda, all animals contain five basic elements: earth, air, fire, water, and space (ether). The distribution of these elements with an individual determines the dominant metabolic body type or dosha (vata, pitta, or kapha). An imbalance of the doshas (caused by improper diet, stress, etc.) creates illness. Treatment is aimed at restoring balance using herbs, diet change, massage, and for humans, meditation and yoga.

Ayurveda for elephants is known as *Hastiayurveda* and was first described in a Sanskrit document written 2000 years ago.[9,40,52] This treatise includes treatments for diarrhea, constipation, parasites, skin diseases (itches, boils, sores, and wounds), abnormalities of temperament, heart disease, and a progressive wasting disease that was probably tuberculosis.[24] Other ancient texts include the Gajasastra and Matangalila (*The Elephant Lore of the Hindus*).[24,40]

Traditional systems, based on ayurveda, but also employing branding, fumigation, and chanting are still used by some native practitioners in Sri Lanka.[50]

## Traditional Chinese Veterinary Medicine (TCVM)

TCVM originated over 2500 years ago and maintains that energy (chi or Qi) flows through channels (meridians) in the body. Disease results when the flow of energy is stagnant or disrupted. Treatment techniques include acupuncture, massage, nutrition, husbandry, surgery, and herbal medications.[46]

**Acupuncture.** *Acupuncture* is the insertion of very fine, sterile needles into points along the meridians corresponding to specific body parts or organs. Electrical or heat moxibustion may also be applied. During acupuncture, energy movement causes blood and Qi to flow smoothly within the body, breaking down blockages and causing effects to mitigate symptoms and stimulate healing (personal communication, Dr. Oh Soon Hock, Singapore Zoo, Singapore, March 2005).

Acupuncture is used in Asia to treat a wide variety of human and animal illnesses. In the U.S. it has been used in humans primarily for chronic pain. It is recognized as a valid treatment modality by the American Veterinary Medical Association (AVMA). Although acupuncture produces analgesia (and anesthesia) a scientific explanation of the underlying mechanism remains elusive. TCVM and acupuncture have been suggested as a useful complement to conventional equine medicine for the treatment of conditions such as arthritic pain, muscle sprains, traumatic paralysis, colic, and diarrhea, although reports are largely anecdotal.[46] Electroacupuncture has been shown to be more effective than phenylbutazone for the treatment of thoracolumbar pain in horses.[71] An elephant acupuncture chart illustrated in a Czech acupuncture book[67] closely resembles depictions of sensitive points in Asian publications.[13,17]

There are only a few reports of the use of acupuncture in elephants. Clinical improvement of chronic front leg lameness in a 56-year-old African female was noted following a 6-month course of acupuncture and herbal therapy, but another chronic case (10-year duration) was ultimately euthanized.[70] Improvement in gastrointestinal function was directly attributed to acupuncture administered over a 4-month period to a 35-year-old Asian elephant. Primary treatment was directed at trunk paralysis secondary to a surgical procedure. The trunk paralysis resolved, but the author hesitated to attribute this to acupuncture alone.[61]

In another case, an Asian elephant sustained an injury during breeding resulting in chronic lameness and inability to flex one foreleg. Response to antiinflammatory drugs given intermittently over a 9-year period diminished and acupuncture treatment was initiated using custom-made needles 150–250 mm in length and 0.5–0.6 mm in diameter (about twice the size of needles used for humans). Prior to treatment the elephant underwent a conditioning period of contact with the acupuncturist to alleviate any apprehension. The initial response to treatment was positive, with a reduction in lameness and renewed ability to flex the foreleg (personal communication, Dr. Oh Soon Hock, Singapore, March 2005) (see Fig. 15.5). For information on TCVM and veterinary acupuncture in the U.S. contact the Chi Institute (www.TCVM.com).

**Homeopathy.** Homeopathy was developed in the 1700s by Samuel Hahnemann, a German physician and chemist. Principles of homeopathy include 1) the law of similars or "like cures like," 2) the selection of a homeopathic remedy that most closely matches the totality of

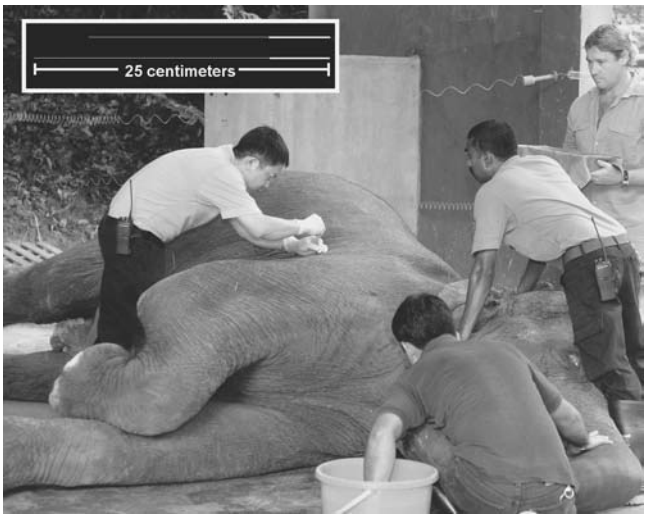


***Figure 15.5.*** *Acupuncture treatment of an elephant at the Singapore Zoo. The fine needles are depicted in the inset (photo courtesy of Dr. Oh Soon Hock, Singapore).*

symptoms, 3) the view that symptoms are the body's attempt to activate its defensive forces to heal itself rather than elements that should be suppressed, 4) the use of minimum doses to stimulate the normal healing process.

Homeopathic remedies are progressively diluted alcohol extract made from plants, animals, or minerals. Critics maintain that most homeopathic remedies are diluted to the extent that the original active ingredient can no longer be detected. Proponents claim a scientific basis in quantum physics.

Homeopathy was widely practiced in the U.S. in the early 1900s and remains an accepted system in Europe, South America, and India. The use of homeopathy in elephant practice has been briefly described.[41] For information on veterinary homeopathy in the U.S., contact The Academy of Veterinary Homeopathy (www.theavh.org).

**Integrative.** Integrative medicine combines the beneficial aspects of multiple disciplines. In its policy position on CAM, the AVMA states "The foremost objective in veterinary medicine is patient welfare. Ideally, sound veterinary medicine is effective, safe, proven, and holistic in that it considers all aspects of the animal patient in the context of its environment. Diagnosis should be based on sound, accepted principles of veterinary medicine" (see www.avma.org). For information on alternative therapies in the U.S. contact the American Holistic Medical Association (www.ahvma.org).

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# 16 Neonatal Care and Hand Rearing

Karen Emanuelson

## INTRODUCTION

Elephant calves are precocious, and a healthy calf born to and cared for by an experienced, healthy elephant mother should not require extensive neonatal care by human caretakers. An elephant calf that is separated from the mother by the adverse health of either animal, or temporary or permanent rejection by the mother (often due to maternal inexperience), *does* require extensive intervention. Successful hand rearing of elephant calves is difficult, even for short periods.

In the past 50 years in North America, approximately 30 African elephant calves have been born at American Association of Zoos and Aquariums (AZA)–accredited institutions, with approximately 17 calves surviving past infancy; in the same period, 87 Asian elephants have been born in AZA-accredited institutions, of which approximately 51 have survived at least past infancy.[23] The survival rate of elephant calves is low compared to other captive megavertebrate species.

The survival rate for elephant calves raised by human caretakers from birth appears to be *very* low, and only a small number have survived to young adulthood.[13] The survival rate of calves reintroduced to the mother after a short period of hand rearing, typically 25 days or less, is much improved.[12] The primary goal of any neonatal health program for elephant calves is to keep the elephant calf with the elephant mother. In cases of forced separation, every effort should be made to reintroduce the calf to the mother.

A significant number of wild-born calves raised by their mothers for a period of time (days, weeks, or months), and then separated (by an event such as death of the mother), have been successfully raised by human caretakers in elephant orphanages in Africa and Asia.[30,32] The chances of survival are greater for older calves and those that have received colostrum. Fostering to another lactating cow is possible; however, a foster cow whose own calf is less than 2 years of age would not be likely to accept a second calf and might not produce sufficient milk for two. Twin calves are rare and typically only one survives.

## PREBIRTH PREPARATION

### Planning

Planning for the birth of an elephant calf should begin a minimum of a year in advance of the expected birth date. An elephant care team composed of animal management and veterinary staff should be formed to develop protocols for dietary supplementation and/or hand raising. The team should carefully review past protocols and experiences with parturition and neonatal elephant calves at their institution if available. They should also consult with personnel with recent experience from other institutions and consult literature and other resources.[5,10,12,20,27,28,29,30]

Supplies should be acquired several months in advance of the anticipated birth date. Some supplies, particularly elephant milk replacer, may require a special order and advanced shipment. A nutritionist and persons with knowledge and experience in hand raising elephant calves should be consulted regarding the appropriate currently recommended milk replacer and feeding regimen.

General supplies for hand rearing an elephant calf should include bovine bottles and nipples, human breast pump, microwave, refrigerator, cooking thermometer, containers and utensils, cell phone, cloth tape measurer, walk-on scales, video camera, still camera, record sheets, notebooks, file folder box, blankets, towels, baby wipes, shavings and straw. Arrangements should be made for 24-hour attendance, along with a list of caretakers and veterinary staff and their contact phone numbers. A nursery area in the elephant barn is necessary including facilities for supplemental heating or cooling.

Medical supplies should include items commonly found in a veterinary clinic such as antibiotics, intravenous fluids, facilities for inhalation and injectable

anesthesia, endotracheal tubes (up to size 10), and equipment for oxygen therapy. If possible a 20–30 mAmp portable radiograph unit should be available.

Elephant plasma (4–6 l) should be collected as much as 6 months prior to the anticipated birth date (from an elephant other than the dam). Colostrum for future use should be collected from the dam after the birth, if the calf has consumed sufficient quantities and the nursing process is not disrupted by this action.

Preparations should include preconditioning the female elephant for possible reintroduction of a rejected calf. If possible, the mother should be desensitized to gentle manipulation of the two mammary glands and nipples, taking care not to massage the glands excessively, particularly near the parturition date, because this could reduce the amount of colostrum available if milk is expressed and could increase the risk of mastitis and mammary gland edema.

A plan for 24-hour care of the calf should be developed prior to birth. Elephant calves quickly bond to their caretakers. The group of caretakers should be a consistent, small group (but large enough to allow for 24-hour scheduling for an extended period); the calf should not be allowed to bond to just one individual (which may lead to extreme separation anxiety on the part of the calf). Initially, two people from the calf care team should be with the calf at all times; the calf should never be left alone.

The elephant management team should develop a plan for the reintroduction of the calf to the dam. The plan should also consider a management program including available facilities, use of outside consultants, the temperament of the dam, veterinary care, and the impact on the staff. Develop a record-keeping system.

## NEONATAL CARE

The gestation period for African and Asian elephants is approximately 20 to 22 months. The average birth weight is 100 to 120 kg (with a range of approximately 53 to 153 kg) for African elephant calves *(Loxodonta africana)* and 90 to 110 kg (with a range of 50 to 148 kg) for Asian elephant calves. The height of elephant calves at birth is approximately 0.9 m (3 ft). A growth chart for elephant calves in North America is available.[23] Healthy calves usually stand, walk, and vocalize within a few minutes to 2 hours after birth and urinate, defecate, and nurse within hours.[31]

The time from birth to the calf's first attempts to suckle ranges from approximately 1 to 6 hours; the time from birth to successful nursing has varied from a few hours to several days (the latter when caretakers have assisted the process and supplemented the calf, as mentioned above). The calf should nurse within the first 6 to 12 hours, in order to receive the full amount of colostrum (2–10 l) during the probable prime absorption period. If the calf does not nurse from the dam within the first 24 hours, supplementation with colostrum and/or plasma should be considered.

### At the Time of Parturition

It is recommended that an apparently healthy calf born to a healthy, multiparous female that has no history of aggression to her calves should not be interfered with by elephant care staff during the initial neonatal period. After successful nursing has occurred and the maternal bonding process is underway, a neonatal examination may be considered if the elephant care team assesses that this will not adversely affect the maternal bond. In all other cases—such as when the mother is nulliparous and has not observed the birth of another female's calf, the mother has a history of aggression toward calves, dystocia has occurred, or the calf is undersized or weak—it is generally recommended that the calf be removed carefully and safely out of the cow's reach immediately after birth, at least for a short period.

The number of elephant care staff in the stall or birthing area during labor should be limited to one or two people with whom the elephant is familiar, and the area should be kept quiet. It is preferred that veterinary staff and other personnel on the elephant calf team watch the labor process by video from an adjacent location.

If it is necessary to separate the calf, it should be carried (usually on a net or canvas sheet, because calves are difficult to grasp effectively) a short distance from the mother so the mother can see, smell, and preferably touch the calf, but not grab it, step on it, or otherwise harm it or the care staff. This period of separation allows the cow to recover from the birth and provides the veterinarian an opportunity to perform a neonatal exam and administer any necessary medications (see discussion below). The elephant care staff may clean the calf and perform initial measurements (such as body weight) while the calf has time to become steady on its feet. When aggression from the mother elephant to the new calf occurs, it tends to occur when the calf struggles to rise and vocalizes. Assisting the calf to avoid struggling may help prevent this aggressive reaction.

If necessary, the cow should be sedated to allow the calf to nurse during this critical period. All activities should be carried out quietly and calmly, with no unnecessary talking, loud noise, or rapid movements by staff in the birth area.

### Initial Care of an Orphaned or Rejected Calf

Assess immediate needs by evaluating respiration, heart rate, and mucous membrane perfusion. Administer emergency therapy including oxygen if needed. If body temperature is less than 36°C (97.5°F), use heat sources such as heat lamps and/or heating blankets to raise the calf's temperature to the normal range of 36–37.2°C (97.5–99°F).

Collect blood samples for a complete blood count and serum chemistry analysis, qualitative IgG test, and

serum electrophoresis. Save extra serum and freeze it. Whole blood in EDTA may be frozen for future DNA testing. Draw blood for aerobic/anaerobic culture if the calf is weak and/or placentitis is present.

Weigh the calf. Administer elephant colostrum if available, 2–10 liters orally by bottle (if there is no possibility of nursing from mother). The calf is unlikely to consume more than 0.5 l in a single feeding. Colostrum should be given within 6 hours and no later than 24 hours after birth. Bovine colostrum may be used if elephant colostrum is not available.

Assess fluid balance. Insert intravenous (IV) catheter if fluid therapy or plasma therapy is indicated. If the calf has not received colostrum, elephant plasma is the preferred fluid. If calf has received colostrum, use LRS and/or plasma.

Perform a thorough physical examination, including the umbilicus. Assess developmental maturity. Examine the placenta to determine whether it is complete. Save samples for culture, sensitivity, and histopathology. Give tetanus prophylaxis, umbilical care, and vitamin E injection.

If possible, make a video of labor, the birth and the immediate postnatal period. Keep detailed records including vital signs, size, time to first attempts to suckle, time of successful nursing, estimated volume of successful nursing in the first 24 hours, time of meconium production, and time of urine production.

Normal vital signs are not well established for neonatal elephant calves, but the body temperature should be 36–37°C (97.5–99.0°F), heart rate 100 to 128 BPM, and respiratory rate 22 BPM. References are available for normal hematologic and serum chemistry values for young elephants.[1,9,23]

### Failure of Passive Transfer (FPT) of Immunoglobulins

It is essential for a newborn calf to receive either colostrum or plasma. Both Asian and African elephant neonates have been reported to consume between 2 and 10 liters of colostrum from their mothers, with nursing beginning as early as 30 minutes after birth. Some calves that are bottle-fed may not drink more than 2 liters the first day of life. In addition to a CBC and serum chemistry panel, total protein, globulins, serum electrophoresis, and a qualitative immunoglobulin test such as the zinc turbidity test should be performed on calves to be hand-reared. Electrophoresis will probably become the most reliable of these tests as more information is gathered.[7]

Elephant plasma should be collected well in advance of the earliest expected calving date. The sterile plasma may be stored at –20°C for 6 months, or at –70° C for 12 months. The donor elephant should preferably not be the mother due to the potential for isoantibodies. The donor should be healthy and herpesvirus negative by whole blood PCR tests, which should be performed with each plasma collection. It is preferable to collect plasma from elephants on site.

The volume of elephant plasma that should be administered to a calf in cases of FPT is not known, but it is likely to be similar to that required for a foal, which is 20 to 80 ml/kg IV over a 2- to 4-day period.[3] A 100 kg elephant calf would require 2 to 8 liters.

A plasma volume of 20 ml/kg/day may be administered to an elephant calf as two intermittent IV boluses, each over 60 minutes, or as a slow drip over more than 2 hours. The first 100 ml should be given slowly and the temperature, heart rate, and respiratory rate monitored. Some calves that have received lower volumes (1.5 liters or less) have succumbed to infections in the post-neonatal period.

Elephant plasma may also be given orally during the first 24 hours after birth (first 6 to 12 hours preferred), but the antibody content is likely to be lower than that found in colostrum; therefore, a larger volume must be given to approach a similar absorption. Colostrum or plasma may have a local protective effect on the intestine even if gastrointestinal absorption is closed.

## MILK

### Milk Supplementation

The calf should not go longer than approximately 19 to 24 hours after birth without consuming milk or some type of milk supplement. Ideally, colostrum should be consumed within 12 hours after birth.

### Milking the Dam

If possible, and if safety concerns allow, the dam should be milked so the calf can receive colostrum and then milked to supplement formula feeding. Milking methods include hand milking, manual human breast pump, and electric human breast pump. Manual milking is similar to that used in goats: Squeeze the teat at the top with the thumb and forefinger and then squeeze the other three fingers in succession; 20 to 30 units of oxytocin may be given intramuscularly approximately 5 minutes before pumping to facilitate milk let down. Frequent milking and the use of oxytocin should dramatically increase the amount of milk collected. One zoo milked their elephant cow every 3 hours and used oxytocin each time, with average collections of 1080 ml per milking during the first week.[5,13]

The breast pump is usually used for approximately 10 to 20 minutes per breast. Rest periods, warm water packs, and massage have been used during milking to increase the volume of milk collected. Milking has also been used to collect samples from a nursing mother for the development and modification of formula.[12]

### Milk Replacers

There is not yet one formula that works successfully for every calf. Analysis of the milk of lactating African and Asian elephants has been reported,[17,20,27] but additional

studies are needed, particularly regarding calcium and fat composition in the formula. Parrott reported the content of African elephant milk on an as-fed basis as 2.3% protein, 4.38% fat, 2.79% lactose, 0.53% ash and 11.32% total solids. On a dry-matter basis, the content should be 20% protein, 39% fat and 24.6% lactose.[27] Mainka reported on the composition of Asian elephant milk at 3.4% protein, 7.6% fat, 0.54% ash, and 19.7% total solids.[17]

The composition of milk varies with the stage of lactation. It would be difficult to compound a formula that is optimal for an elephant calf throughout its nursing period. Consultation with a nutritionist is recommended.

Grober's Elephant Calf Milk Replacer (Grober Nutrition, Cambridge, Ontario, Canada, www.grober.com, 519-622-2500) is one of the most commonly used elephant milk replacers. Nutricia Elephant Calf Milk Replacer has been used in Israel and the Netherlands.[13] Several human infant formulas have also been used to bottle feed calves, including most commonly Wyeth's SMA Goldcap (also known as S26, manufactured in England) or Enfamil.

Grober produces both African and Asian elephant milk replacer, which have been formulated from the analysis of milk collected from lactating females (see Table 16.1). The African formula has 750 kcal/liter, the Asian formula 1215 kcal/liter and Enfamil has 666 kcal/liter. In some cases additional dietary supplementation may be provided. Desiccated coconut and butterfat have been added to increase the fat in the diet.[33,34] However, the supplementation of too much fat could interfere with calcium absorption. The calcium:phosphorus ratio should be 1.5–2.0:1. Vitamin and mineral supplements are commonly used (vitamins B, E, C, and calcium). In many cases rice water and glutinous rice broth have been used when mixing the formula to alleviate diarrhea.

**Table 16.1.** Daily Quantities (Liters/Day) for Different Hand-Raising Formulas (Kinzley 2004)

| Formula | 100 kg Calf | 200 kg Calf |
|---|---|---|
| Grober Asian | 5–6.6 | 13.2–16.5 |
| Grober African | 8–10.7 | 21.3–26.7 |
| Enfamil | 9–12 | 24–30 |

Two examples of formula mixtures used in Southeast Asia are presented in Table 16.2.

Formula should be made up no more than 48 hours in advance and kept refrigerated. Formula that is left at room temperature for more than an hour should be discarded. Hygiene is of utmost importance. After every feeding, bottles should be washed with soapy hot water, thoroughly rinsed (soap left in bottles may cause diarrhea), and dropped in boiling water for several minutes (personal communication, Ware, Phnom Penh, Cambodia, May 2005).

Formula changes should be made slowly by combining formulas and gradually changing from one formula to the other. Abrupt changes tend to cause diarrhea (see discussion below). Calcium supplements may be a cause of constipation; adjust the amount if needed and make increases slowly. A veterinarian should always be consulted if diarrhea persists for more than 1 day; or if constipation occurs; or if there are any other adverse clinical signs, such as lethargy, reduced appetite, or fever.

## NURSING TECHNIQUES

A normal calf should be allowed to attempt to find the mother's mammary gland on its own for the first several hours after birth. After approximately 4 to 6 hours, if the calf needs assistance finding the nipple and safety concerns allow, offer the calf a clean finger to suckle on while guiding it toward the nipple. Another method is

**Table 16.2.** Selected Formulas Successfully Used in Asia for Asian Calves

| Elephant | Ingredients | Amount | Directions |
|---|---|---|---|
| Calf #1Indonesia[a] | Lactogen 2 (Nestle's) | 60 grams | Fed daily between 10–15% of body weight. Initially fed q 2–3 hrs. At 2 months night feedings reduced. Used bovine calf nipple. |
| | Brown Rice Porridge | 60 Grams | |
| | Sucrose | 20 Grams | |
| | Bone powder | 35 grams (qs to 1 liter with water) | |
| Calf #2[b] | Dumex full cream milk powder (East Asiatic Company Ltd.) | 125 grams | Add ingredients to 1 liter of (Dumex) milk. Lactase Enzyme drops added to the milk 24 hrs prior to use. Final mixture contained 20% solids, 750 kcals/l, 3.5% protein, 7.25% fat, 5.78% CHO, 1.03% Calcium:calcium/phosphorus ratio of 1.8/1. |
| | Corn oil | 50 grams | |
| | Overcooked rice | 75 grams | |
| | Osteoform (Vet-a-mix Australia) | 2 grams | |

[a]Manansang 1997.
[b]Personal communication, Dave Ware, Phnom Penh, Cambodia, May 2005.

to use an IV line attached to a fluid bag containing milk, with the end of the IV line attached to the caretakers' fingers so the calf may be more easily led to the dams' teat. An undersized calf may need a stool to stand on in order to reach the nipple.

When bottle feeding is necessary, a bovine calf nipple is used, with the nipple opening slightly enlarged to allow a steady drip when inverted. Initially the milk or milk replacer should be offered to the calf at a lukewarm (tepid) temperature, approximately 29.4°C (85°F); the formula should always be tested by placing a drop on the inside of the wrist to make sure it is not too hot. Do not microwave formula; this could create "hot spots." When the calf is older, the milk may not need to be heated.

Some calves may struggle to find a comfortable nursing position for bottle feeding and will not nurse well until they do. They appear to need to have their trunks up against something. Some calves need intimate contact with the keeper and will come to rest their trunk at the underarm, face, neck, etc. Some caretakers have had success with hanging a piece of canvas for the calf to push up against to nurse. It may be comforting to place a blanket over the calf, including covering the ears, so just the face is visible. Thin blankets or sheets may be used in warmer weather.

Newborn elephants often have difficulty lying down, and have been observed shuffling around the enclosure until they stop and begin falling asleep. At some point they just collapse or tip over. They may also need help in getting up. Large canvas sleeping cushions, straw bedding, or whole bales of straw should be provided for the calf to lean against.

### Feeding Amounts

It is estimated that calves weighing 100 kg (220 lbs) should receive approximately 6,000 to 8,000 kcal per day and calves weighing 200 kg (440 lbs) should receive 16,000 to 20,000 kcal per day. The volumes of Grober Asian and African formula and Enfamil required to achieve these caloric needs are presented in Table 16.1. Overfeeding is probably not a concern in very young (neonatal) elephant calves. The calf should gain 0.5kg–1.4 kg (1–3 lbs) per day, averaging 0.9 kg (2 lbs) of weight gain per day during the first year of life.

### Feeding Schedules

Calves should be fed on demand initially. The feeding interval for young calves is generally every 1 to 2 hours, around the clock. At 3 months of age, the frequency of nighttime feedings may be gradually reduced, depending upon the demands and growth rate of the calf. Some caretakers begin to shift gradually to feeding once every 3 hours during the daytime at approximately 3 to 6 months of age; or, the calf may continue to be fed on demand. At 1 year of age, bottle feeding should be every 4 hours during the hours of daylight, with one to two night feeds if the calf is hungry. At approximately 15 months of age, an elephant calf should be eating solid foods in sizable quantities, and the milk may be decreased to three bottles a day.

### Solid Foods

Hand-raised calves experiment with solid foods at an early age, but they appear to develop normal feeding habits much more slowly than mother-raised calves. Mother-raised calves use their trunks to smell and take food from the mouth of their mothers, learning what is desirable to eat. Small amounts of solid food such as hay and other adult elephant feeds may be offered to the calf beginning at 1 or 2 months of age. Hand-raised calves often are very interested in tasting foods being eaten by their caretakers. Caretakers might be able generate more interest in appropriate foods by pretending to eat them with the calf (do not share foods directly, to prevent the direct sharing of oral flora). Elephant calves generally do not begin eating significant amounts of solid food until at least 6 to 9 months of age, with a real increase in the second year of life.[2,32,33,34] They are milk dependent for 2 years, but solid foods are gradually increased in the second year, and milk is decreased.

### Nursery Location

Planning should include the development of a nursery that is safe from the adult elephants but allows visual, auditory, and olfactory contact. Chain link works well as a barrier for the calf but the smaller (1 in × 1 in) links are necessary to prevent the calf from putting its trunk through the fence. Calves are sensitive to cold temperatures. Generally, the nursery area should be kept at about 18°C (65°F), but a very young or ill calf many require a warmer ambient temperature. Blankets and heavy bedding may help to offset cold temperatures. The space should have a drain and be able to be sprayed clean with a hose; calves will produce large volumes of urine and feces. A layer of shavings covered by a deep straw bedding works well to insulate the calf and to absorb urine and feces between cleaning. A service area with electricity, water, and storage space should be nearby.

### Socializing with Other Elephants and Reintroduction

When the mother is still present, reintroduction of the neonatal calf is of paramount importance.[12] If this cannot be accomplished, continued attempts should be made for the calf to spend significant time (most of the day and night) near the mother with a protective barrier between them if necessary. This may benefit the calf and the mother, and may increase the chances of the mother forming a bond with another calf in the future. Elephants that are hand-raised tend to prefer human companionship to that of other elephants, unless socialization is encouraged early.

In at least two cases a calf was introduced to a foster mother early in life[13] (also personal communication, J. Flanagan, Houston, Texas, April 2005). This allowed the calves to socialize normally while still being bottle-fed. It is critical that the calf is allowed every opportunity to socialize with other elephants. The nursery should be in close proximity to the elephant stalls so the calf can hear, smell, and see the other elephants. If possible, regular periods of interaction with the other elephants should be planned. The elephant management team should develop a plan and timetable for integrating the calf into the herd as soon as possible.

### Manners and Training

It may be very difficult to control the movement of young calves. They may not follow well; a bout of exuberance can send them running off in any direction, and they may be resistant to attempts to direct or stop them. The environment that they are kept in should be "baby-proof" so that they are not in danger if their caretaker loses control of them. Although pushing and herding may be effective, grabbing and pulling seems to trigger an escape instinct and the calf will fight to get away. As they get older they become more reliable followers and respond to their name and simple directional commands. A firm "no" seems to be effective in teaching them what they cannot do, but avoiding problem situations is often the most practical approach in calf interactions.

Calves exhibit normal butting and charging play behavior at an early age. Calves should not be allowed to interact with humans in any way that would be unacceptable for an adult elephant. Having many toys available like plastic garbage cans, boat buoys, and hanging objects will make it easy to divert the calf to a toy when a play bout begins. The calf will learn to direct play behavior toward inanimate objects. Conditioning the calf to allow examinations and behaviors that are important for its welfare and husbandry needs, using repetition and positive reinforcement, is important. In many cases keepers have been able to "capture" simple behaviors such as "lie down," "trunk up," "move up," "back up," etc., by naming and reinforcing the behaviors when they occur.

### Play and Exercise

It is important for normal and healthy development that the calf receives regular exercise. Calves in the wild would be walking miles each day. Even in a captive situation a mother-raised calf would spend more time walking as it followed its mother than a calf raised in a nursery situation. If possible the calf should be allowed to follow the keepers through their cleaning routine. Exercise should also come in the form of play sessions. Healthy calves should regularly have energetic play sessions, which may be solicited with favorite and novel toys.

## PHYSICAL AND BEHAVIORAL DEVELOPMENT

Physical development of hand-raised calves should follow closely that of mother-raised calves. Monthly body measurements, photographs, and videotape of the calf will allow close monitoring of the calf's growth and development. The calf should be weighed daily. If a hand-raised calf is not able to spend an adequate amount of time with other elephants, normal behaviors may be absent or slow to develop. The caretakers may encourage some behaviors, such as dusting, eating solid foods, mud wallowing, swimming, and play behaviors. Further study is needed to determine the effect of hand raising on the calf's communication and social skill after it is integrated into a herd.

## WEANING

In the wild, elephant calves are weaned at 3 to 5 years of age, generally around the time of the birth of the female's next calf.[14,15,16,22] The weaning process tends to be gradual. This natural process of weaning should be imitated in captivity if possible, in cases of mother-reared calves in a healthy group. Following the goal of developing naturalistic and healthy social elephant groups, which are likely to enjoy better health and reproductive success, calves should not be forcibly removed from their mothers, particularly until the age at which separation (primarily in males) occurs in the wild. In hand-reared calves, the socialization of the calf with other elephants on the premises may be facilitated by weaning. The weaning process should begin at approximately 12 to 14 months of age with the gradual decrease of milk and gradual increase of solid foods, including cereal added to the milk formula. The calf should not be completely weaned before 2 years of age and generally should continue some nursing until at least 3 years of age. Elephant calves in one orphanage in Africa may continue some nursing until the age of 5 years.[32]

## COMMON MEDICAL PROBLEMS

### Diarrhea

Loose stool in a variety of colors may be "normal" for formula-fed infants. Stool that is unusually odorous, separated into chunks, or liquid is abnormal. The frequency of stool production, and the appearance of the stool that is considered normal for one particular calf, is helpful for determining the extent of diarrhea when it occurs. When diarrhea is severe or accompanied by other clinical signs such as lethargy, weakness, reduced appetite, colic, or dehydration, diagnostic evaluation as well as treatment is necessary. In addition to a thorough physical examination, recommended diagnostic tests include a complete blood count; serum chemistry analysis; urinal-

ysis; aerobic and anaerobic fecal culture for potential pathogens, including *Salmonella, Clostridium, Campylobacter,* enterotoxigenic *Escherichia coli, Pseudomonas,* and others; fecal examination for parasites; and a fecal cytology smear. Additional diagnostic tests to consider include herpesvirus PCR and antibody testing if available, blood culture, fecal exam for cryptosporidia and giardia, fecal electron microscopy or other evaluations for viruses, and abdominal radiographs.

Blood collection, and also positioning for x-rays, may be stressful for elephant calves, so the necessity for these should be carefully considered.

**Treatment.** Changes in formula are used to manage mild diarrhea that occurs without any additional clinical signs. Options are 1) to dilute the formula 25% to 50% for 1 to 3 days; 2) to discontinue the formula and substitute water, electrolyte solution such as Pedialyte, rice water, or rice milk for 1 day; 3) alternate each formula feed with a feed of electrolyte solution for 1 to 3 days; or 4) change to a different formula. Other therapies may be used, such as kaopectate orally, antibiotic therapy, intravenous fluid therapy, and antiparasitic therapy when appropriate.

## Constipation

Constipation may occur following a stressful event or an abrupt diet change. Signs include listlessness, anorexia, abdominal contractions with no defecation (straining), absence of defecation, and rubbing hindquarters against the walls.

Therapy may include warm water enemas (may be necessary daily) antibiotics, corticosteroids, and vitamin B12 if the calf is weak. Oral cathartics such as mineral oil should be used with caution in young animals, because they may cause further abdominal discomfort and/or diarrhea.

## Metabolic Bone Disease/Rickets

Metabolic bone disease may result from an imbalanced calcium:phosphorus ratio or intestinal malabsorption. Published and anecdotal reports indicate that fractures typically occur in Asian calves, at 8 or 9 months of age, and may involve multiple limbs. Euthanasia is often the result[4,11,21,25,7] (also personal communication, J. Flanagan, 2005). Not all cases have been attributed to a nutritional etiology; however, calcium imbalance has been suspected in most cases. Calcium metabolism is complex and absorption may be influenced by the level and type of fat in the diet. Further research is needed, and dietary vitamin D levels also need to be established. The hindlimbs of the calf should be evaluated radiographically at approximately 6, 7, and 8 months of age. The calf should perform mild exercise daily and the calf should have access to sunlight at least three times weekly, although daily exposure is preferred. Treatment of metabolic bone disease involves correcting the dietary imbalance, possible vitamin D injections, monitoring calcium levels, rest, and consideration of additional therapies used in other species.

## Herpesvirus Infection

Herpesvirus is a significant cause of neonatal mortality in elephants. Thirty-two cases of herpesvirus infection have occurred in elephants worldwide between 1983 and 2005, all but two in Asian elephants, mostly in very young animals.[6] The disease is acute to peracute, and it is usually rapidly fatal. The infection is more likely to occur in Asian elephants that are housed with or near African elephants and in elephants that live or have lived on premises where a case has occurred previously. Stress may be a causative factor in the development of the disease. See Chapter 11 for more details. It is recommended that hand-reared and mother-reared calves be trained to allow a physical examination, particularly of the oral cavity; the oral cavity should be inspected with a flashlight twice daily in young elephants. Cyanosis, petecchial hemorrhages, edema of the tongue, and generalized lethargy are hallmarks of herpesvirus infection. An adequate supply of the antiviral drug Famciclovir should be kept on site. See further information in Chapter 15.

## Sunburn

Elephants are susceptible to sunburn, especially on the head. Ensure that adequate shade is available during outdoor time. Sunscreen has been used on calves; however, effectiveness is uncertain and there could be some potential for allergy, so covering the calf with sheets and keeping the calf in the shade may be better options. Treat sunburn with a soothing cream, such as vitamin E cream, and restrict access to sunlight until healed.

## Skin Dryness

Dry skin has been noted in hand-raised calves, which may cause a marked pruritis. Treat by applying a mixture of lanolin and mineral oil (454 g [1 lb] lanolin added to 3.8 l [1 gal] mineral oil]) to the calf's skin one to three times weekly after gently bathing the calf with warm water. It may be advisable to test the calf for allergy to the mixture by applying a small amount to the skin the first time it is used. Be careful to avoid sunburn when the skin is oiled.

## Umbilical Infection

Umbilical infections have been reported in elephants, including one fatality. The umbilical sheath may be mistaken for an umbilical cord. The cord (umbilical artery and veins) actually retracts inside the sheath into the abdomen. When disinfecting the umbilical sheath, use a syringe to deliver the disinfectant into the open umbilical sheath, because just dipping the structure may not be adequate.

Serious umbilical infection may be more likely in a

calf that is immunocompromised by FPT, stress, or other factors.

Diagnostic evaluation includes aerobic and anaerobic culture and sensitivity. Treatment in mild cases consists of cleansing, gentle debridement, and antiseptic flush with dilute povidone iodine or chlorhexidine solution once or twice daily. In more advanced cases, or particularly in cases of FPT, add topical antibiotic irrigation and systemic broad-spectrum antibiotic therapy.

### Trauma

An infant that has been rejected may have received traumatic wounds from the mother, other elephants, or by birth accident. Diagnostic evaluations include physical examination, aerobic and anaerobic culture and sensitivity of any infected wounds, CBC, chemistries, and radiology. A 30–40 mA portable X-ray machine can image the extremities, but higher mA may be necessary for radiographs of the thorax or abdomen. Anesthesia may be required to X-ray a calf that is not depressed or weak. Treatment may include daily wound care, topical or systemic antibiotic therapy, analgesics, surgery, rest, and supportive care.

## THERAPY AND PREVENTIVE MEDICINE

### Fluid Therapy

The adult elephant fluid maintenance requirement is 30 to 60 ml/kg/day.[7] The infant requirement is likely to be higher. Active disease and fluid losses may increase fluid requirement to 2 to 4 times maintenance. Subcutaneous fluid administration is not a preferred route due to limited SQ space, although it may be used with some success in the more dehydrated animals. Fluid is well absorbed by the oral and rectal route, and these should be used whenever possible.

Subcutaneous or intravenous fluid therapy may be used when marked dehydration is present, especially if the calf is anorexic. It is difficult to maintain an IV catheter in an infant for continuous infusion. One option is to give fluid by intermittent IV bolus. As a guideline, no IV bolus should exceed 40 ml/kg, 20 ml/kg being preferred; and the fluids should be given as slowly as possible, over a 60-minute period. Sedation should be considered if the calf is extremely stressed by restraint for IV fluid administration. See Chapter 15 for more details. IV catheterization may be accomplished with an 18-gauge or 20-gauge catheter placed in the medial saphenous vein or ear vein. Sloughing may occur with the injection of medications other than fluids into the vasculature of the ear. It may be difficult to thread an over-the-needle catheter through the skin on the hindlimb. Butterfly catheters may also be used but are difficult to keep in place, even for short periods. Use buffered 2% lidocaine infused into the tissue over the vein for catheter placement. A topical local anesthetic cream such as Emla cream may be useful. Suture or apply surgical adhesive to help keep the catheter in place.

Lactated Ringers is a suitable fluid solution for most cases. Do not add potassium chloride if the fluids are given as a rapid IV bolus. Glucose may be added to make a 2.5% or 5% solution if the blood glucose is reduced below 60–80 mg/dl.

### Antibiotic Therapy

Antibiotics used in treatment of elephant calves include ampicillin, amoxicillin, penicillin G procaine, and ceftiofur in less severe infections; amikacin or gentocin may be considered in more severe cases, when hydration is maintained; if possible, serum levels are measured. Avoid fluoroquinolones and tetracyclines in young growing animals. Antibiotic therapy may be critical in neonates, especially in cases of FPT. No pharmacokinetic studies have been done comparing juvenile to adult elephants. Few studies have included captive African elephants in particular, and the dosages could vary as compared to Asian elephants. Intramuscular or intravenous routes of administration are preferred to oral, due to the potential for iatrogenic diarrhea. Repeated IV doses in a calf would be difficult due to stress unless the calf is moribund.

## PREVENTIVE MEDICINE

A preventive medicine program for the elephant calf (or calves) should be developed by the veterinary staff and coordinated with the entire elephant care team. The program should include daily visual checks (including oral exam) by animal care staff and frequent examinations by the veterinarian (at least weekly in hand-reared neonates). See Chapter 7 for more details.

### Vaccinations

Tetanus toxoid, a vaccination for *Clostridium tetani,* has been given to adults, subadults, and neonates at multiple zoological institutions. Give 1 ml IM, with the first dose at 3 months of age, and the second dose at 4 months of age. Consider an initial dose as early as the first day if the calf has not received colostrum.

Tetanus antitoxin has been administered in adults and could be considered for use in a neonate with lesions likely to become contaminated with C*lostridium*. However, fatal anaphylaxis has occurred following administration of tetanus antitoxin to horses, sheep, and cattle. Uncommon vaccinations such as those for other clostridial diseases, West Nile virus, encephalomyocarditis virus, and rabies may be indicated in endemic areas. Signs of and treatment for adverse reactions to vaccinations should be discussed with caregivers by the veterinarian. Treatment for anaphylaxis is discussed in Chapter 15.

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# 17 Multisystem Disorders

Murray E. Fowler

## STRESS

Stress is the cumulative response of an animal to interaction with its environment via receptors[11] or, as another author defines it, "stress is the biological response elicited when an animal perceives a threat to its homeostasis."[26] The threat is a *stressor* (stress-producing factor), and it is important to recognize that a psychological perception of a threat may be as important as the response to a physical stressor. The biological responses brought about by stress are adaptive, directed at coping with environmental change, and every animal is subject to stress, whether free-ranging or in captivity. Intense or prolonged stimulation may induce detrimental responses (*distress*).[26]

Species vary in their perception of a threat and how they process the information received to evoke a physiologic response. It is not possible to use any single laboratory parameter to determine the stress status of an animal.

Little significant research has been conducted of stress in elephants, so the following thoughts are based on research in other animals. We cannot be complacent and assume that elephants don't become distressed.

A stressor is any stimulus that elicits a biological response when perceived by an animal. A listing of some of the potential stressors acting on elephants may direct attention to consideration of these important factors when handling elephants. *Somatic stressors* (stimulation of the physical senses) include temperature changes; strange sights; unfamiliar sounds, touches, or odors; thirst; and hunger.

*Psychological stressors* include anxiety, fright, terror, anger, rage, and frustration. Closely allied are *behavioral stressors,* including overcrowding, lack of social contact, unfamiliar surroundings, transport, and lack of appropriate foods. Miscellaneous stressors include malnutrition, toxins, parasites, infectious agents, burns, surgery, and drugs.

It is becoming more important to recognize that stimulation of visual and auditory senses has a marked bearing on accumulative stress. Modern interpretation makes no distinction between specific and nonspecific responses because there is marked species variation in how organisms process and act upon stimuli.[27] There may even be varying responses from an individual, depending on which stimuli are acting on it at a given time and the experience, hierarchical status, nutrition status, or history of a previous adaptation to the stimulus.[4,24,27]

### Body Response to Stress Stimulation

The central nervous system (CNS) receives messages from receptors, processes the information, and initiates a biological response through one or more of the following pathways: behavior, autonomic nervous system, neuroendocrine system, or the immune system.[16,24]

Animals respond in appropriate ways to stimulation of specific receptors. For instance, when cold receptors are stimulated, the body experiences a sensation of coolness. Various somatic and behavioral changes occur that conserve heat and stimulate increased heat production. The animal is adjusting to a new situation (*homeostatic accommodation*). If heat is the stressor, the animal tries to take steps to cool itself.

The autonomic nervous system deals with short-term stress responses (flight or fight scenario); however, any tissue innervated by autonomic nerves may be affected (such as increased peristalsis). The autonomic nervous system is seldom a factor in distress because the duration of stimulation is short.

The neuroendocrine system is a major pathway for the development of distress. Often this pathway is thought to be the hypothalamic-pituitary-adrenocortical (HPA) pathway. However, modern research has conclusively demonstrated that all systems modulated by the hypothalamic-pituitary axis may be affected (growth, reproduction, immunity, metabolism, behavior).[24,25,27]

Individual animals and species vary in the primary pathway utilized to cope with change. The pathways

used by the elephant are unknown. However, continuous adrenal cortex stimulation and excessive production of cortisol elicit many adverse metabolic responses. Psychological as well as physical changes may occur. The clinical syndromes of adrenocortical stimulation have been identified in some species (human, dog, horse, laboratory animals). There is much still to learn about the effects of hypercorticism in elephants. However, the basic biologic effects of cortisol should be understood.[11,14,16]

Protein catabolism and lipolysis contribute to the pool for glyconeogenesis. Slight-to-moderate hyperglycemia has a diuretic effect, producing polyuria and polydipsia. Prolonged hyperglycemia stimulates the beta cells of the pancreas to produce more insulin.

Cortisol reduces the heat, pain, and swelling associated with the inflammatory response, an effect useful in the treatment of many diseases. The antiinflammatory action of cortisol is brought about by reducing capillary endothelial swelling, thus diminishing capillary permeability. Additionally, capillary blood flow is decreased by the action of cortisol. Both of these actions are helpful in shock therapy.

The integrity of lysosomal membranes is enhanced by cortisol. Under such circumstances, bacteria and other particulate matter are engulfed by phagocytes, but hydrolytic enzymes (which would destroy the organisms) are not released from the lysosomes.

Within a few hours of a cortisol stress response there is a reduction in the number of circulating lymphocytes (50% or greater). Lymphocyte levels return to normal within 24 to 48 hours following cessation of stress. The effect of stress on the total leukocyte count varies with the species and depends on the normal relative leukocyte distribution. Species with normally high percentages of lymphocytes, such as mice, rabbits, chickens, and cattle, respond with a lymphopenia and neutrophilia and a decrease in total leukocytes. Dogs, cats, horses, and human beings, having relatively low lymphocyte counts, respond with an increase in leukocytes.[11]

Elephants generally have a slightly higher percentage of lymphocytes than neutrophils, but the numbers are close enough that it is difficult to identify a stress hemogram in an elephant. Eosinophil production decreases in response to elevated levels of cortisol. Eosinophil production is directly related to histamine production, such as occurs in the event of tissue injury or allergic reactions. Cortisol neutralizes histamines and inhibits regranulation of mast cells, thus further reducing histamine production. The elevated production of cortisol during stress results in eosinopenia. Catecholamines also cause eosinopenia; thus emotional stress may elicit a stress hemogram. In addition, cortisol stimulates increased production of circulating erythrocytes. Serum calcium levels decrease through inhibition of calcium absorption from the gastrointestinal tract.

Stress ulceration of the gastrointestinal system is a well-known syndrome in humans, rats, and marine mammals. Whether stress is a factor in elephant ulcers is unknown, but studies have determined the basic effect of cortisol on the digestive system.[11,14] Most of the studies have been performed on humans and laboratory animals, and as there may be significant species differences, direct extrapolation is unwise. The pathogenesis of gastric stress ulcers in humans and marine mammals is multifactorial. Hypercortisolism causes hypersecretion of acid and digestive enzymes. A duodenal reflux introduces substances from the duodenum into the stomach (*lysolecithin*) that reduce the effectiveness of the mucous membrane barrier. A third factor is vasoconstriction of the vasculature of the stomach, which in turn causes local hypoxia and a deficiency of adenosine triphosphate. These also contribute to the reduction of the mucous membrane barrier. Whether these factors are operating in the elephant is unknown, but should be considered.

Catecholamines (epinephrine) contribute to the production of gastric secretions, so stimuli mediated via the sympathetic nervous system (fear, anxiety, frustration, anger) may have a potential effect on ulcerogenesis.

## Reproduction

Intense and prolonged stressor stimulation has been found to be detrimental to the normal reproductive cycle. Acute response to stress, such as restraint or transportation, has been found to inhibit ovulation in livestock.[25,27] Stressor stimulation may prevent ovulation by inhibition of the preovulatory secretion of luteinizing hormone (LH). Another mechanism may be via the HPA axis, which may inhibit a corticotropin-releasing hormone that is essential for the production of gonadotropin-releasing hormone (GnRH), which, in turn, is essential for ovulation to occur.

## Metabolism

Metabolic changes associated with stress may shift resources that are needed for basic functions such as growth, especially during critical growth stages. Suppression of thyroid function occurs as a result of neuroendocrine derangement.

## Immunity

Several mechanisms may act on the immune system to inhibit normal immunocompetence.[2,7,27] Interference with DNA synthesis causes atrophy of lymphoid tissue throughout the body. Cell-mediated immune responses are diminished, an effect that may interfere with appropriate response to vaccination and tuberculin-testing programs. Lymphopenia decreases the number of leukocytes available to combat infection.

## Diagnosis of Stress

Signs vary with the pathway stimulated. Because few studies have been conducted on stress in elephants, it is

unlikely that a diagnosis can be made on the basis of signs. No single laboratory determination is definitive as a diagnostic tool for distress, but plasma cortisol levels are commonly used as an indication of stress. However, just the collection of blood from an elephant for analysis may cause an increase in plasma cortisol. Steroid levels in feces and urine are used as indicators in some wild animals, and salivary cortisol has been used as a noninvasive method to monitor stress. In one captive Asian elephant cow, salivary cortisol predictably rose from a baseline of 6.17 ± 1.43 nmol/l to 31.8 nmol/l following introduction into a new herd.[8] Plasma cortisol is only one measure of stress. Of equal importance to this study was that the stressor stimulation was of short duration (2 days), after which cortisol levels returned to baseline levels. Thus the introduction into a new herd did not produce distress in this elephant.

Stress response protein (SRP) profiling is a novel technique currently under investigation in elephants as a method to detect chronic physiologic stress and disease. It is based on measuring levels of 40 stress response proteins using immunohistochemical staining and image analysis. Stress-related alterations in SRP profiles are similar among the mammalian species studied thus far and appear capable of differentiating healthy animals from those with disease or physiological stress.[1]

### Pathology

The lesions produced by distress (harmful stress) are difficult to document. Pathologists often negate a diagnosis of death caused by stress. Many of the effects of stress are functional, leaving no definitive lesion to mark their presence. Nonetheless, it is known that tissues and organs are weakened by prolonged insult, lowering resistance to disease. Classic lesions are lymphoid tissue atrophy, adrenal cortical hyperplasia, and gastrointestinal ulceration. Though the actual cause of death may be pneumonia, parasitism, or starvation, stress may have paved the way for development of these terminal ailments.

### Summation

Stress is ever present in both free-ranging and captive elephants. It is crucial that stress remains at levels that are beneficial to the elephant and do not rise to become distress, which is detrimental to elephant well being. Veterinarians providing health care for elephants should consider stress as a contributory factor in specific diseases. Husbandry practices should be evaluated and correction of those that may be harmful recommended. Elephants are social animals. Isolation for therapy or recuperation may be counterproductive. Malnutrition is a stressor, as are repeated and prolonged restraint episodes.

The stress response mechanisms employed by elephants are unknown, which should make stress research a high priority in elephants. More detailed information about stress may be obtained from the references.

## THERMAL STRESS

Free-ranging elephants are found in tropical, subtropical, semiarid, or desert environments.[17,30] Although there are anecdotal reports of elephants in extreme temperatures, modern heat balance investigations suggest that the ability of elephants to acclimate to extreme temperatures may be limited.

### Thermoregulation (*This Section by Susan Mikota*)

The low surface-to-volume ratio and apparent absence of sweat glands (except for interdigital glands) has raised numerous questions regarding how elephants maintain thermal balance. Investigations are ongoing to define the precise physiological mechanisms involved.

Early studies suggested that the ear, with its comparatively large surface-to-volume ratio, extensive blood supply, and frequent motion was primarily responsible for heat dissipation.[5,28,33,39,40] By flapping its ears, the African elephant adds 13–20% additional surface area for heat; Asian elephants can increase their radiative heat loss area by only 7–10% because of their much smaller ears. This is the only species that can increase or decrease the radiative area for heat loss (personal communication, Dr. Vaughan Langman, Shreveport, Louisiana, April 2005); however, heat transfer across the ears may represent only about 8% of the total heat loss.[38] Although the motion of elephant ears seems almost constant, African elephants don't actually flap their ears until the temperature is nearly 25°C (77°F) (unpublished data, Langman, April 2005).

Transdermal evaporative water loss, facilitated by normal behaviors such as bathing, spraying, and mud wallowing has also been suggested.[15] The wrinkles, characteristic of elephant skin, may adsorb water and facilitate its movement over the surface of the skin, thereby helping to mitigate evaporative heat losses. It has been suggested that the deeper sculpturing of the skin of the African elephant may function more efficiently in this capacity, providing an obvious adaptive advantage to living in a savannah habitat with frequent exposure to direct sun.[23] Evidence from recent thermographic studies, however, does not support a constant heat loss from the surface due to sculpturing and insensible water loss from the skin (unpublished data, Langman, April 2005).

The advent of thermographic technology has provided a tool to further evaluate heat transfer in elephants (see Chapter 13). In one study of two elephants, convective, radiant, and conductive heat transfer were measured at six major anatomical sites. At a temperature of 12.6°C (54.7°F), convection and radiation accounted for 86% of the total heat loss.[38]

Thermal conductance is a measure of the amount of heat leaving from an insulation barrier. High thermal conductance is indicative of a low degree of insulation.

In the same way, low insulation values predict a high thermal conductance. Large cats and hoofstock living in climates with seasonal temperature changes increase and decrease their seasonal conductance accordingly. During cold weather, conductance is reduced by adding insulation (hair or body fat).

A recent 3-year study comparing thermal conductance in African elephants and tigers over a wide range of temperatures has demonstrated 1) a higher thermal conductance of elephants especially as ambient temperatures increased (temperatures at which tigers would likely pant to dissipate heat), and 2) an inability of elephants to insulate or adjust to extremes in ambient temperature compared to tigers and mountain goats (unpublished data, Langman, April 2005). Thus while elephants are able to vasoconstrict and vasodilate the blood vessels in their skin (as humans do) they do not add insulation in the winter (to decrease thermal conductance) or lose insulation in the summer (to increase thermal conductance).

Obligatory heterotherms are characterized by having adult body weights of 1000 kg or greater and no evaporative mechanism for losing either internal or external heat loads. Without evaporative mechanisms to maintain a heat balance these megafauna use the enormous thermal inertia inherent in their body mass for heat storage.[19]

Empirical evidence that elephants are able to allow their core body temperature to elevate and dissipate the excess heat later when ambient temperatures are lower, usually at night, was first reported by Elder.[9] This "heat sink" is also utilized by the dromedary camel.

In a natural environment in the wild, the stored heat is lost from the skin's surface by long-wave radiation to the night sky, bringing the body temperature to 33C–34°C (91.4–93.2°F) by the early morning. In a captive environment, however, this heat loss process may be affected by the structures in which elephants are housed or transported.

Heat balance and thermal regulation are critical to health and well being and should be considered when designing exhibits or transport vehicles for elephants[22] (see also Langman[18,20,21]). Although previous anecdotal reports have indicated that elephants can adapt to wide temperature ranges, these recent data suggest that their abilities to acclimate may actually be quite limited. Further, because elephants appear to use thermal conductance as one of their main ways to dissipate heat, elephants under thermal stress may not demonstrate a rise in core body temperature until the stress is severe.

## Hyperthermia—Heat Exhaustion, Heat Stroke, Sunstroke, Heat Stress (*Remainder of Chapter by Murray Fowler*)

Hyperthermia has not been recognized as an important problem in adult elephants, but it does occur.[19,30] It may be more important in calves that are maintained in a captive environment that is less than optimal for elephants.

**Etiology.** The predisposing factors for development of hyperthermia in elephants include prolonged, high environmental temperatures and humidity, muscular exertion, fever, dehydration, mycotoxins that inhibit thermoregulation, and drugs (chemical restraint agents) that depress thermoregulation.[12,32] Activities that may contribute to the production of body heat include using elephants for trekking, elephant rides, or the aggressive behavior of bulls in musth. Elephants being transported may be at risk, but one study found no thermal stress associated with transporting circus elephants.[37] Frequent monitoring of the core body temperature is important during prolonged restraint.

The normal core body temperature of adult elephants ranges from 36–37°C (96.8–98.6°F).[3,5,6,9,37] Normal temperature of neonates may be a degree higher than that of the adults.

The degree of hyperthermia and the effects of hyperthermia on organs and tissues may vary according to the duration of exposure to excessive heat and humidity and the presence of other conditions, such as metabolic acidosis, cardiovascular dysfunction, or chronic disease.

**Effects on organ systems.** It is often assumed that when a hyperthermic animal has been cooled, all organ systems begin functioning again at their normal capacity. That may be true if the heat stress has been of short duration and moderate intensity. If heat stress has been severe or prolonged, many residual effects may alter organ function and even cause death long after the core body temperature has returned to normal. A veterinarian must understand the effects of heat stress on organ systems to make an accurate diagnosis, treat a case effectively, and give a prognosis.

Central nervous system. The CNS is highly sensitive to hyperthermia. Effects on the CNS may be initiated by direct effects of heat, causing coagulative necrosis of neurons, or by secondary factors, such as hypotension, causing cerebral hypoxia, or electrolyte alterations, resulting in neurotransmission dysfunction. Lesions in the CNS may also be caused by hyperthermic damage to the cardiovascular and hemic systems (hemorrhage, disseminated intravascular clotting [DIC]). Signs exhibited are determined by the area of the CNS damaged, but generally there is decreased mental function and ultimately convulsions. Damage to thermoregulatory centers may predispose animals to relapses or subsequently increased sensitivity to heat.

A frequently overlooked but serious consequence of heat stress in a pregnant female is fetal CNS damage, resulting in various congenital anomalies or even death of the fetus. Congenital CNS defects associated with prenatal prolonged hyperthermia in humans and other ani-

mals include exencephaly, anencephaly, encephalocele, microencephaly, spina bifida, hydrocephaly, and neurogenic arthrogryposis. These anomalies are the result of excessive heat acting on the embryonic cells of the CNS at a crucial time. In humans, the critical time is between 40 and 44 days following fertilization. The crucial period in elephants is unknown.

Reproductive system. Heat stress may have a marked effect on the adult female, including diminished receptivity and anestrous. During pregnancy, the more profound effects are seen as fetal damage, including inhibition of embryonic cleavage and implantation, initiation of teratogenesis, and abortion.

General effects on the fetus may result in reduced birth weight, which may be caused by placental retardation. Fetal effects have been noted in other species when the core body temperature of the dam rises above 40.1°C (104.2°F) for prolonged periods. Hyperthermic effects on an embryo are dependent on the degree of hyperthermia, duration of hyperthermia, and the stage of development of the embryo.

Abortion may be the result of placental necrosis, direct effects on the fetus causing death (microvascular leakage, edema, hemorrhage), or, in near-term fetuses, a stress response causing elevated cortisol levels.

In the male, excessive heat is spermicidal at the primary spermatocyte stage. Elephants are more susceptible than most mammals because the testicles are intraabdominal with less possibility of anatomical cooling.

Respiratory system. A 1°C (1.8°F) rise in the body temperature increases the requirement for oxygen by 10% to maintain normal function of the energy systems of the body. If the body temperature rises to 41°C (105.8°F), the respiratory system is no longer able to supply sufficient oxygen by normal respiration. Heat stress causes tachypnea and respiratory acidosis.

Digestive system. Signs of colic may be seen in a heat-stressed elephant. Elevation of the core body temperature initiates a shift in the blood supply from the viscera to the skin and ears. Decreased blood flow to the stomach and intestine causes decreased digestive function. Gastrointestinal motility is decreased.

Hypoxia of hepatocytes and decreased hepatic function results from decreased blood flow to the liver. In severe cases there is a failure of production of elements in the coagulation cascade. Persistent low intensity hyperthermia may cause decreased digestive function, which in turn may cause poor growth rates in juveniles and poor appetite and less efficiency in feed utilization in adults.

Cardiovascular system. Hyperthermia causes dilation of peripheral arterioles and a shift of blood from the viscera to the skin and ears. Heart rate is increased and central venous pressure is decreased, along with a relative decrease in blood volume (potential for hypotension and hypovolemic shock).

Hematopoietic system. Hyperthermia causes hemoconcentration, electrolyte imbalances, increased fragility of erythrocytes, leucocytosis, and metabolic acidosis. Platelet counts are decreased. Effects on the coagulation cascade may be profound and lethal. Prothrombin time is increased, and there is an increased consumption of coagulation factors and fibrin split factors, resulting in hemorrhage and, potentially, disseminated intravascular coagulation (DIC).

Urinary system. Hypovolemia may result in decreased glomerular filtration and loss of kidney function (prerenal uremia), followed eventually by renal shutdown, possibly complicated by DIC. Generalized hemolysis overloads the kidney with hemoglobin, which exacerbates any kidney malfunction or may be the direct cause of kidney malfunction.

**Sequence of events during hyperthermia.** As may be ascertained from the foregoing discussion, many changes in organ systems affect the clinical signs manifested. Diagnostic tests may be employed to assess organ system function and potential for residual effects that may complicate recovery or result in the death of the animal some days later. Following is the probable sequence of physiological changes and signs developed during a severe hyperthermic episode in an elephant (Fowler 1995b):

1. Elevation of the core body temperature
2. Accelerated heart rate
3. Increased respiratory rate
4. Sweating around the eyes and toenails
5. Hemoconcentration
6. Body fluid shift from viscera and muscle to the skin and ears
7. Decreased glomerular filtration
8. Dehydration
9. Decreased central venous pressure
10. Effects on the CNS, including cerebral hypoxia and coagulative necrosis
11. Effects on the embryo and fetus
12. Coagulation defects (disseminated intravascular coagulation)
13. Other organ system damage

**Clinical signs.** *Heat stress, heat exhaustion, heatstroke,* and *sunstroke* are terms used to describe slightly different clinical syndromes associated with an elephant's inability to dissipate excessive body heat. The principle sign is an elevated core body temperature. If working, an elephant becomes fatigued, which progresses to depression, rapid breathing, ears drooped, trunk becoming

somewhat flaccid, incoordination, ataxia, trembling, recumbency, delirium, convulsions, and death.[10] Other signs may be related to thermal effects on various organ systems, including dyspnea, accelerated heart rate, and sweating around the eyelids and at the base of the toenails. Dehydration and decreased urine output may be noticed.

Possible sequelae to a hyperthermic episode include teratogenesis, aspermia, disrupted reproductive cycle, renal shutdown, hepatic insufficiency, DIC, and mental impairment.

**Differential diagnosis.** Primary hyperthermia must be differentiated from an infectious disease producing a fever.

**Necropsy.** Gross lesions suggestive of death from hyperthermia include hemorrhages and DIC. Coagulation necrosis of the neurons of the CNS and necrosis of the liver and kidneys would be characteristic histopathologic findings.

**Therapy.** Successful resolution of hyperthermia requires early recognition, rapid cooling, supplying supplemental oxygen, fluid administration to deal with hypovolemia, and correction of acidosis. A cold water enema should be given immediately. Crushed ice may be packed around the ears and head. Make sure the elephant is out of direct sunlight. Stretch a tarpaulin over the elephant if necessary. The patient should be monitored through sequential hemograms and serum chemistry profiles measuring hemoconcentration, electrolytes, pH, platelets, and the coagulation cascade elements.

A basic maintenance fluid requirement for a healthy resting adult elephant is unknown, but extrapolating from equine requirements it should be approximately 60 ml/kg/day. Calves may require up to 120 ml/kg/day. Depending on the degree of dehydration, two to five times the basal requirement may be necessary. Normal saline or lactated Ringer's may be used to supply fluid requirements. Electrolyte imbalance must be dealt with according to serum chemistry findings. Acidosis may be controlled by administration of a sodium bicarbonate solution intravenously.

**Prevention.** Managers must provide shade and water for cooling.[36] A study quantifying the value of shade structures for elephants determined that shade may be beneficial in reducing the heat load, but in extreme conditions, a controlled environment may be necessary.[22] Restraint procedures should be avoided on warm days.

## Sunburn

Elephants may become sunburned if exposed to direct solar radiation for a prolonged period. Signs, diagnosis, and management are as for other animals.

## Hypothermia

**Predisposing factors.** Predisposing factors include exposure to subzero temperatures in northern areas of the world, where winter nighttime temperatures may dip to −40°C (−40°F). When combined with wind chill, the effective temperature may be −73°C (−100°F). Without shelter, elephants will have difficulty coping with such severe cold.

Neonates are particularly susceptible to hypothermia because they have poorly developed thermoregulatory mechanisms and a higher metabolic rate. Their relatively greater proportion of skin surface to body mass allows for rapid dissipation of heat. Neonates have a diminished shivering reflex. Even adult elephants under anesthesia or in shock are prime candidates for hypothermia if in a cold ambient environment.

Insufficient food intake reduces metabolic heat production. Restricted muscular activity prevents heat generation.

**Signs.** A low core body temperature is the primary sign. Other signs include depression progressing to coma. In contrast to hyperthermia, the hypothermic elephant may live for hours.

A decrease in body temperature is accompanied by a decrease in cardiac output, heart rate, blood pressure, and glomerular filtration rate. Blood viscosity and hematocrit levels increase. Signs noted with body temperatures below 30°C (86°F) include slow and shallow breathing, metabolic acidosis, "sludging" in the microcirculation, ventricular arrhythmias leading to fibrillation, and coagulation disorders.[12]

**Therapy.** A warm-water enema is highly effective, but the ability to monitor the body temperature is temporarily lost. A hair dryer may be helpful in warming a neonate. For an adult, a construction forced-air heater would be required. Covering the animal with blankets will help conserve heat, but if the body temperature is below 32.3°C (90°F), metabolic heat production is proportionately reduced, and endogenous rewarming is slowed.

Intravenous infusions of warm saline are effective. Surgical exposure of a suitable vein may be necessary to effect IV administration because of vasoconstriction. Circulating water–type heating pads are effective in preventing hypothermia in neonates during surgery and in treating accidental hypothermia, but electric heating pads have caused skin burns and sloughs. Hypothermic and shock patients normally suffer from skin vasoconstriction and exhibit a reduced ability to carry heat away from the skin. Caution is necessary when heat is applied directly to the skin. The temperature between the skin and the pad should be measured and kept below 42°C.[12]

**Prevention.** Shelter from wind and rain should be provided. Insulated and heated barns may be required in particularly harsh, cold climates.

High-quality feed should be provided, including concentrates if elephants are accustomed to eating them. Water must be available.

### Frostbite

The elephant is capable of constricting the arterial blood supply to the ear during cold weather. If the cold stress is intense and prolonged, freezing of the peripheral edges of the ear may occur, just as it does in many species of mammals. Clinical signs, diagnosis, and management are as in livestock species.

## EXERTIONAL STRESS

Exertional stress is a complex alteration of metabolic processes that may cause peracute-lethal acid base and electrolyte imbalance or more commonly produces acute-to-delayed necrosis of skeletal and cardiac muscle.[13,34] The syndromes produced are usually associated with capture and restraint of wild ruminants, but they are also seen in horses, cattle, sheep, dogs, primates (human and nonhuman), elephants, and birds. These syndromes are not reported in the elephant literature, but are observed in recently captured elephants in Southeast Asia (personal communication, Dr. Susan Mikota, Waveland, Mississippi, April 2005).

Names applied to the syndromes vary with the predisposing factors and clinical signs exhibited and include *capture myopathy, stress myopathy, overstraining disease, muscular dystrophy, capture disease, degenerative polymyopathy, ataxic myoglobinuria, idiopathic muscle necrosis* and *white muscle disease*.

### Predisposing Factors

Predisposing factors include fear, anxiety, overexertion, repeated handling, failure to allow an exhausted animal to rest before transporting, and constant muscle tensions such as may occur in protracted alarm reactions. A variety of stressors may function together or individually to precipitate development of any of the syndromes.

### Etiology

An elephant responds to a restraint or capture episode by releasing catecholamines, which in turn, initiates complex neurohormonal responses. Muscle activity generates heat, and when muscle glycogen becomes depleted, anaerobic muscle metabolism produces lactic acid. Muscle ischemia caused by hypoxia or the restraint practices compounds the problem.

As with other forms of stress, exertional stress is protective initially, but if the exertion is prolonged and intense, distress occurs, evidenced by acidosis, electrolyte imbalance, hypoxia, hyperthermia, pulmonary edema, and muscle necrosis. Serum biochemical alterations associated with exertional myopathy are discussed in Chapter 25.

### Syndromes

**Peracute mortality (capture shock).** This syndrome may occur within 15 minutes of a stress episode or 6 hours later. Signs include severe depression, shallow rapid breathing, hyperthermia, tachycardia, circulatory collapse, and death.

**Muscle necrosis (ataxic myoglobinuria).** Signs of acute muscle necrosis caused by elevated lactic acid levels may occur in 3–6 hours or several days later, depending on the degree of muscle damage. Signs of muscle necrosis include ataxia, paresis, paralysis, myoglobinuria, and elevated serum enzymes associated with muscle necrosis. If heart muscle becomes necrosed, acute death may ensue.

Excessive levels of myoglobin released from the muscle cells are excreted through the kidney, but may impact in the renal tubules and cause tubular necrosis resulting in renal failure.

**Ruptured muscle syndrome.** Heavily used muscles are most often necrosed. If sufficient fibers are damaged, muscle integrity is destroyed and the muscle may rupture. The gastrocnemius muscle is frequently involved, resulting in hyperflexion of the hock. In the elephant, the hock is an integral part of the foot, so hock flexion may not be as evident as in other species. This sign may be obscured if the animal is recumbent.

**Delayed effects.** An exertional stress episode may be mild enough that apparent recovery takes place, even with some necrosis of skeletal or cardiac muscle fibers. If another stress episode occurs within a few days, before healing is complete, the musculature is more sensitive to adverse effects. This is particularly dangerous if cardiac muscle is involved. Acute cardiac collapse may occur.

### Necropsy

Lesions may be minimal in peracute mortality because the adverse effects are biochemical. However, pulmonary congestion and edema may be seen. Muscle necrosis may be noticed as light grayish streaks in affected muscles.

### Management

Prevention must be paramount. All restraint procedures should be well planned and carried out as quickly as possible by an experienced team. Chemical restraint may minimize the effects of stress, but capture myopathy may occur during chemical restraint.

Monitor body temperature and cool hyperthermic animals quickly. Supplemental oxygen insufflation may be helpful in captive elephant immobilization. If blood pH determination is not available and if the course of the capture or restraint episode indicates excessive exertion, administer an initial dose of sodium

bicarbonate (4–6 mEq/kg) and repeat the dose if signs indicate a need.

After muscle necrosis occurs there is no specific therapy and the prognosis will depend on the degree of necrosis.

Although the administration of selenium and vitamin E has been useful in the prevention of white muscle disease in bovine calves, it has no value in therapy of capture myopathy.

## ELECTROCUTION

Electrocution is not a common disorder of elephants, but accidental and malicious electrical shock has been reported.[10,35] Electrocution has also been used to euthanize rogue elephants.

In the United States, standard electricity is supplied as alternating current, 120 volts and 60 Hz (cycles per second). 230 V power may be installed in homes and commercial institutions (zoos) for powering clothes driers, cookstoves, arc welders and other equipment requiring heavy power usage.

### Etiology

Elephants may receive an electrical shock either from a lightning strike or contact with a power line.[10] A lightning strike may be a direct hit, or the charge may spread through the roots of a tree or wet soil to an elephant some distance away. Oak trees are prone to receive lightning strikes. An electrical charge caused by lightning may also follow a metal fence or envelop a building. Elephants are prime targets for lightning strikes because of their size.

Elephants may be exposed to bare high tension transmission lines that are not strung out of reach of an elephant or that fall during storms when trees are toppled. Farmers trying to protect their crops from marauding elephants have strung wires around their crops and have used standard household voltage, which is sufficient to kill any animal, instead of electrical fence voltage.

### Clinical Signs

**Lightning strike.** Elephants, like people, may survive a lightning strike. Unless the strike is witnessed, it may be difficult to make a diagnosis, but the following signs may be noted: burns on the skin, depression, blindness, nystagmus, paralysis, and temporary unconsciousness. Characteristically, there is a history of an electrical storm and evidence of lightning damage in the area. A single lightning strike may kill more than one animal, especially if a group has congregated under a tree for shelter. Lightning may kill so quickly that there is still feed in the mouth.

**Electrical current.** Elephants are sensitive to electricity. Commercial electric fencing is often used as a barrier to protect trees and shrubs from browsing. Usually, after the first contact, elephants avoid an electric fence. Commercial electric fencing is controlled by an energizer (transformer) that converts alternating current to direct current at an appropriate voltage and pulsation. The energizer may be powered by standard electric current in the country where it is used. An energizer may also be powered by batteries or solar panels.

Legal electric fences are being used in Asia and Africa to prevent marauding elephants from reaching valuable crops and structures. Some of the fences are highly effective and others are not, primarily based on design and consistent maintenance.[35] Individual elephants may breach an electric fence by knocking down posts or rushing the fence. Males may break wires using their tusks, which are poor conductors of electricity, or by stepping on wires with their slipper, which is also a poor conductor.[35]

It is important to realize that a solid contact with the standard 110 V current or household and barn electrical service is sufficient to kill animals. The amount of damage done depends on how well the elephant is grounded (wet surfaces are more dangerous) and the duration of the contact with the current.

Electrocution usually shocks the heart, causing cardiac arrest or ventricular fibrillation. Electrical current often follows the course of nerves or blood vessels and may destroy those vital structures by the heat generated. Electrical current stimulates the nervous system and causes the muscles to contract, the strongest muscles overpowering the weaker ones. A serious problem in people who touch electrical wires is that the shock causes the hand to close and grasp the wire, thus continuing contact with the current. An electrician always touches a suspect wire with the back of his hand, so that if the wire is hot, the shock will pull the hand away from the wire. Consider what would happen if an elephant grasps a wire with its trunk.

### Management of Electrocution

Always make certain that the victim is disconnected from any electrical current**.** If the victim is still touching a downed power line, quickly determine whether it is possible to turn off the current. If not, call the emergency line of the local utility company. Do not attempt to move the wires away with a dry stick, a piece of wood, or plastic pipe, or cut the wire even with a pair of lineman's side cutters that have handles covered with rubber or plastic. If the voltage is too high, insulation may not be sufficient to prevent electrocution of the rescuer.

Cardiopulmonary resuscitation is the only effective first aid measure for any animal. Unfortunately the elephant's size, anatomy, and physiology make this impossible to perform on an elephant.

## OBESITY

Many of the elephants in North America are considered to be obese. Exercise is minimal and feed readily avail-

able. Overfeeding is not only costly, but obese animals are more likely to be infertile and develop hyperthermia more easily than those of normal weight.

There is no question that some elephants become obese more readily than others. Elephant managers must adjust feed intake to body condition. See further discussion in Chapter 6.

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# 18 Integument System

Susan K. Mikota

## UNIQUE ANATOMY

The integument system of the elephant is comprised of skin, hair, nails, glands, and the soles of the feet. The integument forms a protective environmental barrier, functions in temperature regulation and waste excretion, and may be considered the largest sensory organ of the elephant. The term *pachyderm* (which includes elephants, rhinoceros, and hippopotamus) literally means "thick skin."

### Skin

The skin is comprised of two layers: the external, nonvascular epidermis and the underlying dermis composed of collagen and containing nerves and blood vessels. Skin thickness varies considerably over the body. The skin on the medial pinnae and at the oral and rectal mucocutaneous junctions is thin. Thicker skin covers the head, legs, dorsum, and trunk. Actual measurements vary from 1.8 mm on the medial surface of the ear to 1.0 cm on the medial side of the leg and 3.2 cm on the dorsum.[67] The skin overlying the lateral thigh muscles of the hindlimb and the triceps muscle of the forelimb (common sites for injections) may be 2.5 cm or more. The skin is usually thinner on the forelimb than the rear.[51]

Histologically, the epidermal–dermal junction in most mammals is characterized by simple papillae or groups of papillae. In the elephant, however, complex polygonal columns ("studs") are found. In some areas these may be as high as 1 cm, adding considerable strength to areas such as the forehead and in contrast to areas (such as the cheek) where the flat epithelium is only 1 mm thick.[32]

The color of the skin is typically gray but may appear brown or rust colored depending on the color of the soil used for mud or dust baths. White elephants, revered in many Asian countries, are not true albinos. These elephants have lighter skin, hair, and nail color, and other specific characteristics.[9,27] Although rare, true albino elephants have been reported.[34] Depigmentation is common in Asian elephants; large depigmented patches on the ears, trunk, face, or abdomen are distinguishing characteristics of the Sri Lankan subspecies (*Elephas maximus maximus*).[68] Elephants can ripple their skin to shake off insects using the expansive underlying panniculus carnosus muscle.[22] This ability is not unique to elephants and is commonly observed in other mammals such as horses.

### Hair

Fetal and newborn elephants have more hair than adults and Asian elephants are more hairy than Africans. Body hairs are black or grayish-brown, bristle-like, and sparsely distributed. Hair is most apparent around the eyes, ears, genitalia, and on the chin and tail. Under light microscopy scales may be seen (as in other mammals) and cross-section reveals concentric circles.[6,67] The gross hair structure of Asian and African elephants does not differ from that of the woolly mammoth, *Mammuthus primigenius*.[77]

Tactile or sinus hairs (vibrissae or whiskers), found primarily on the face, are a mammalian feature. These hair follicles have a rich blood and nerve supply contained within a collagenous capsule.[47] In the elephant, the unique sensory innervation of the trunk tip (comprised of vibrissae in the skin, vellus vibrissae beneath the skin's surface, and corpuscular receptors in the superficial dermis) provides an anatomical basis for the well-documented sensitivity of this area.[59]

### Glands

Most mammals possess sebaceous (sebum-secreting and associated with hair follicles) and/or sudoriferous (sweat) glands. Sweat glands may be apocrine or eccrine. Apocrine sweat glands are large, specialized glands found in the axilla, anogenital region, and groin of humans and in numerous locations in hairy mammals.[39] Apocrine glands are involved in thermoregulation and may produce pheromones. Eccrine glands empty di-

rectly onto the surface of the skin and function in thermoregulation by evaporative cooling. The cutaneous glands of the elephant include the temporal, mammary, sebaceous, and interdigital.

Temporal glands, unique to elephants, are paired modified apocrine sweat glands. They are located beneath the skin midway between the lateral canthus of the eye and the external auditory canal in the temporal fossa on either side of the face. They are present in African and Asian elephants of both sexes.[21,23] The temporal gland may be the site of abscess formation (Fig. 18.1).

The glands are comprised of numerous lobules divided by fibrous connective tissue trabeculae emanating from a thick fibrous tissue capsule. Large arteries, veins, nerve bundles, and a series of interlobular ducts follow the trabeculae.[18,21,28] Temporal gland abscesses should be probed carefully because of the underlying vasculature. See Figure 14.5 in Color Section. Tubulo-alveolar secretory end pieces in the parenchyma empty into a series of connecting ducts that join to a single main duct opening to the surface via a pore in the temporal fossa.[1,80] Fibroblasts, macrophages, plasma cells, and occasional mast cells are seen histologically.[21] The temporal gland produces a secretion that is intimately involved in chemical communication. This function is discussed in Chapter 32.

The paired mammary glands are located in the pectoral region between the forelegs. Numerous large and small ducts with fibromuscular walls and highly vascular intertubular tissue comprise the parenchyma. The nipples are almost circular in shape.[43,67] The histo- and cytophysiology of the lactating mammary gland have been described.[81,82]

Sebaceous glands have been reported.[43,72,75] The existence of true sweat glands has long been argued based on the observation of damp skin under the harnesses of working elephants.[22,70] Histological evidence has been lacking[43,67,73] until 2001 when interdigital glands, similar to human eccrine sweat glands, were demonstrated in Asian elephants.[39] These glands appear to be the source of moisture noted in the interdigital spaces and on the cuticles. Their function remains unclear.

### Nails

Nails are discussed in Chapter 20.

## UNIQUE PHYSIOLOGY

### Sensitive Points

The skin is richly innervated and possesses sensitive points. *Hastyayurveda,* the ancient Indian text, states that there are 107 sensitive points—44 on the limbs, 3 on the lower abdomen, 9 on the chest, 14 on the back, 12 on the neck, and 25 on the head.[14] Twenty-seven points are mentioned in certain Burmese writings, however only 15 correspond to those used in India.[25] Pressure applied at these points causes various learned actions that have been used to communicate with, train, and control elephants.

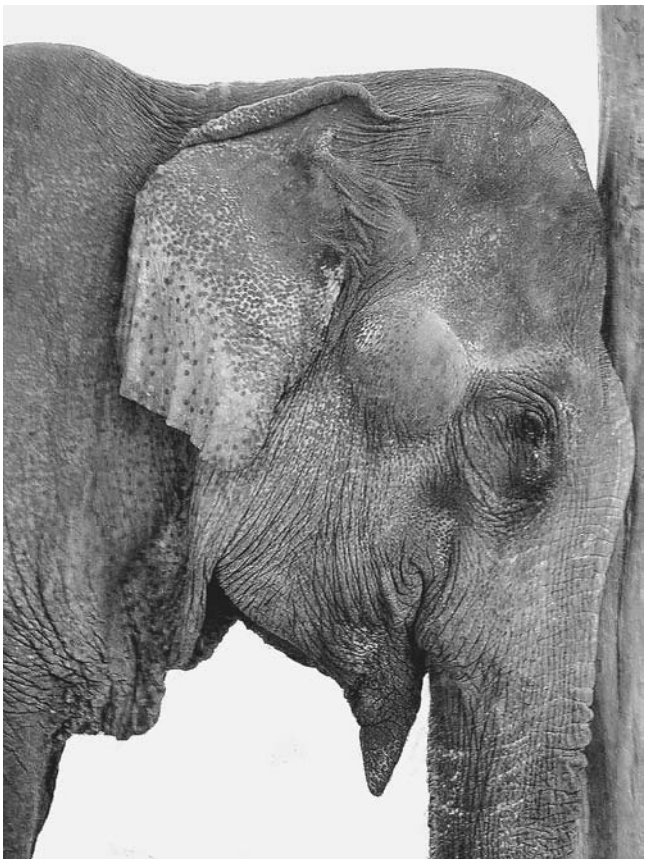

***Figure 18.1.*** *Temporal gland abscess in an Asian elephant. Abscesses in this area should be probed carefully (photo by Hank Hammatt).*

### Thermoregulation

Thermoregulation is discussed with multisystemic disorders in Chapter 17.

## SKIN CARE

Wild elephants bathe frequently and cover themselves with dust or mud that likely serves to maintain healthy skin and ward off parasites.[60,69] Flyswatters fashioned from branches assist in pest control.[29] Disorders of the integument are common among captive elephants, and skin care is an important aspect of husbandry. In Asia it is recommended that at least 75–90 minutes a day be spent on this important task. Vigorous scrubbing is encouraged to remove dirt from skin folds, especially where ectoparasites occur (behind the ears, base of tusks, tail, and axilla). Coconut husk or burnt brick (commonly called *jhama* in India) may be used for scrubbing. The application of fly repellent around the nails and the base of the tusks is advised in elephants kept in forest areas.[36] In zoological settings, stiff brushes and pressure sprayers may be used.[52] Exhibit design

should include natural bathing, wallowing, and dusting areas to support skin health. A design for an artificial scratching post is available.[79]

## DISEASES AND DISORDERS

Healthy skin is dark, pliable, of uniform temperature, and free of major wounds. Skin disorders may be of infectious (parasitic, bacterial, viral, fungal) or noninfectious (nutritional, sunburn, wounds) etiology. Disorders of the integument accounted for one-third of all medical events reported in a retrospective study of elephants in North America.[44] A study of 140 domesticated elephants in Sri Lanka revealed a high prevalence of wounds (44%), punctures (54%), and ulcerated feet (69%), attributed to poor husbandry and improper handling.[26] Pododermatitis is discussed in Chapter 20.

### External Parasites

External parasites of elephants include lice, fleas, ticks, flies, and mosquitoes. These are discussed below. See details in Chapter 12.

### Dermatitis

Dermatitis (inflammation of the skin) can result from extremes of temperature, mechanical or chemical irritation, malnutrition, and infectious or parasitic disease. Signs may include pruritis, hyperemia, swelling, excoriation, and ulceration.

**Parasitic dermatitis.** Parasitic dermatitis occurs most commonly in Asia and Africa. Severely pruritic lesions due to *Stephanofilaria* are seen on the feet, shoulder, or abdomen.[2,4,11,16] Histopathology reveals hyperkeratosis, parakeratosis, acanthosis, and microabscesses. Microfilaria may be detected on deep skin scraping. Treatment with 8% metrifonate ointment in a base of Indian herbs or vaseline is effective following daily treatment for 15–22 days.[76]

Cutaneous filariasis caused by *Indofilaria* spp. results in large, 1–2 cm nodules on the underside of the abdomen or lateral limbs (Fig. 18.2). After 1–2 days, the nodules rupture and ooze blood at 10-second intervals for 30 minutes. Treatment with anthiomaline (50 ml/2000 kg) subcutaneously weekly for 8 weeks is effective.[13,15]

Dipterous larva may produce small nodular eruptions from which larva can be expressed. When the larva is mature, the fly exits, leaving a small hole in the skin. Secondary infections may develop that are usually bacterial, but mycotic dermatitis has also been recorded.[12]

**Superficial dermatitis.** Culture and/or biopsy may be required to establish a definitive diagnosis of superficial skin lesions. *Staphylococcus*, *Streptococcus,* and *Candida* are the most likely organisms to be isolated.[44] Treatment may include topical antibiotic or nonantibiotic preparations, lavage, steroids, or debridement, depending on the lesion and underlying etiology. Purulent trunk dermatitis in an aggressive Asian elephant was successfully treated with topical antibiotic sprays and a long-acting penicillin administered at periodic intervals under sedation. An autogenous vaccine was given when Candida was isolated and the diet was supplemented with probiotics and vitamins.[78]

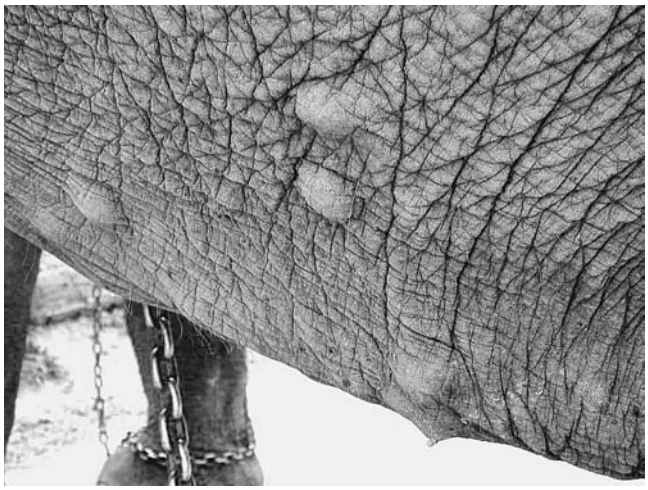

***Figure 18.2.*** *Raised lesions characteristic of cutaneous filariasis (photo by Susan Mikota).*

**Sunburn and other burns.** Despite their thick skin, sunburn may occur in elephants exposed to strong sunlight. Erythematous lesions may occur on the head, forehead, or back.[57] Acepromazine in combination with xylazine has been associated with photosensitization resulting in sunburn.[13,17] Burns may occur during fires or from contact with hot objects or caustic chemicals. In many cases the skin will slough. See management under "Wounds, Abrasions, and Lacerations" (below). There is a photograph of an elephant with sunburn in the Myanmar section of Chapter 35 (Fig. 35.12).

**Nutritional dermatitis.** In one case, vesiculobullae above the toenail and hyperkeratosis on the elbows and tail were attributed to a primary zinc deficiency causing a secondary immune deficiency. Lesions resolved within 8 weeks after increasing dietary zinc from 21.56 to 53.6 mg/kg feed on a dry matter basis.[65]

**Decubital ulcers (pressure sores).** Pressure sores may occur on the hips, elbows, or other pressure-sensitive areas with prolonged contact on inappropriate surfaces. Pressure may compromise circulation, causing tissue damage, deep ulceration, and open wounds. Lesions may be dry and painful or swollen and irritated. Treatment should be aimed at identifying and correcting any underlying etiology. Elephants may lie down for long

periods due to pain, foot problems, or other ailments. Exposure to rough, cold, or unhygienic substrates may be causative or may aggravate an underlying problem.

Providing rubber mats, wooden pallets, or straw may reduce the pressure to affected areas. Consideration should be given to installing heated floors in new facilities, particularly in colder climates. A variety of topical agents have been used for symptomatic treatment with varying success. Oral antiinflammatory/pain medications such as ibuprofen may be helpful. Biostimulation using low-level infrared light therapy (Equi-Light, 2100 S. Dayton, Denver, Colorado 80231, USA, http://www.equi-light.com/main.htm) has shown promise to stimulate granulation tissue. Light-producing diodes are attached to a flexible pad that connects to a power source. The pads are placed over the wound and taped to the skin. Treatment periods may last 30–45 minutes and can be administered daily or less frequently depending on the severity of the lesion. In one case, a pressure sore measuring 10 × 7 cm was reduced to 3.4 × 1.5 cm and developed a healthy granulation bed following 3 months of therapy.[24] Transdermal oxygen therapy has been used for pressure sores in humans and may have application in elephants (see http://www.ogenix.net/).

**Dry skin.** Dry skin may result from lack of humidity or insufficient bathing. Treatment with oil-based preparations such as vegetable oil, mineral oil, lanolin, or baby oil may be helpful. Dry crusty skin seems to be especially problematic in African elephants.

## Wounds, Abrasions, and Lacerations

Both free-ranging and captive elephants have ample opportunity to sustain injuries to the skin. Puncture wounds may result from sharp objects (thorns, nails) or from the improper use of guide devices (ankus or hook). Leg chains applied too tightly may cause abrasions, lacerations or even degloving (avulsion of the skin from the underlying structures). Used fire hose placed over chain can help to protect against such injuries. The tail is particularly prone to injury; bite wounds from other elephants and hydraulic door accidents are common causes.

The goal of wound management is to prevent further contamination, remove debris, debride dead tissue, provide drainage, and create an environment conducive to the formation of a healthy vascular bed.[40] Most superficial wounds will resolve quickly if kept clean and allowed to heal by secondary intention.[37] Wounds can be flushed with sterile saline (or boiled water if sterile solutions are not available), dilute povidone iodine or dilute chlorhexidine. Suturing is not generally indicated as dehiscence is common. Even large skin lesions will heal if a healthy granulation bed is able to form. It appears that multifocal beds of fibroangiomatosis originating from around hair follicles eventually coalesce to achieve healing in a novel manner in these large wounds (personal communication, Dr. Gregory Bossart, Ft. Pierce, Florida, USA, March 2005). See Figure 18.3 (Color Section). A variety of topical agents can be applied; however, keeping the wound clean and devoid of necrotic tissue is most important. Topical antiseptics and antibiotics are discussed in Chapter 15. The administration of tetanus toxoid should be considered for deeper wounds, although data on dose and effectiveness in elephants is lacking. A good Internet resource for information on the treatment of human and veterinary wounds is www.worldwidewounds.com.

**Snares, gunshot wounds, and land mines.** These types of injuries often involve underlying soft tissue, have an increased likelihood of infection, and require more aggressive treatment. Snare injuries may occur on the trunk or limbs. Successful reattachment of an amputated trunk is unlikely, but elephants are remarkably adaptive and can learn to eat even after the loss of the distal 20 cm of trunk.[7] The author has seen an elephant with a >180° encircling laceration of the distal trunk who also successfully compensated for his injury (Fig. 18.4). Elephants in Asia that survive land mine accidents often sustain dramatic injuries requiring months of intensive treatment (see Fig. 18.5, Color Section).

## Abscesses and Necrotizing Fasciitis

Abscesses form readily in elephants and may result from penetrating injuries, tusk wounds, or injections administered by hand or remote injection. Abscesses may appear as hard or fluctuant swellings beneath the skin and may be warm to the touch. Detection is not always obvious as abscesses may spread beneath and undermine the skin rather than rupturing externally.[64] Needle aspiration to confirm a diagnosis may be unrewarding unless a large bore needle is used, because purulent material can be quite thick. Warm compresses may help to draw the abscess to the surface. Abscesses should be incised at their ventral border to establish drainage. Large abscesses may require a second more dorsal incision to facilitate lavage.

Purulent exudate should be cultured if possible and antibiotics selected based on sensitivity results. Local treatment may be sufficient, but large abscesses encountered in unhygienic field situations may require systemic antibiotic therapy.[45]

The tendency of abscesses to spread beneath the skin may have serious consequences. Infection may progress (often undetected) for months, resulting in necrotizing fasciitis, sepsis, and death. This is most likely to occur in situations where initial diagnosis and treatment have been delayed.[45] See Figure 18.6; see also Figure 18.7 (Color Section). Care should be taken to clean the skin thoroughly when giving intramuscular injections. Wounds created when elephants are darted in the field should be cleaned, and a prophylactic systemic antibiotic administered. Antibiotic paste preparations de-

***Figure 18.4.*** *Healed snare injury of 10 years' duration. Elephants often compensate remarkably for severe injuries. This bull was able to eat and drink adequately and was in good body condition (photo by Hank Hammatt).*

signed for intramammary use in cattle may be instilled into the dart wound site after flushing.

## Segmental Gangrene

Cellulitis, necrosis, and tissue sloughing of the ears may result from the extravascular administration of drugs such as phenylbuatzone.[46] Dilution in a large volume of fluid and administration via a catheter may be wise precautions when administering irritating drugs using auricular veins. Frostbite may also cause ear sloughing. Topical treatment to prevent infection is indicated.

## Viral Diseases

**Poxvirus.** Pox is discussed in detail in Chapter 11. Cutaneous lesions consist of 1–3 cm vesicular eruptions on the tongue, oral mucous membranes, trunk tip, eyelids, and perianal and perivulvar skin.[38] Clear, bloody or purulent fluid may be released if vesicles rupture. The lesions become crusty and unpigmented scars may form over a period of days to weeks. Vesicular lesions may coalesce, ulcerate, and become generalized in immunocompromised elephants. Pox can be fatal, especially if infection extends into the corium of the nails.[38]

***Figure 18.6.*** *External swelling caused by a dart abscess in a wild Asian elephant (photo by Hank Hammatt).*

**Papillomavirus.** Papillomas (warts) may occur on the skin, the oral or nasal mucosa, and at the oral mucocutaneous junction. An indirect peroxidase-antiperoxidase test or virus isolation confirms infection. Papillomas appear to be transmissible between elephants. Homeopathic treatment and autogenous vaccination may evoke a temporary remission, but removal by electro-cautery or excision appears to be more effective long term.[31,33,74]

**Herpesvirus.** A herpesviruslike infection was implicated in multifocal nodular fibrous lesions occurring on the trunk, head, eyelids, limbs, and sides of wild-caught African elephants imported to the U.S. in the early 1980s.[33] Preserved samples from these papillomatous lesions subsequently demonstrated herpesvirus sequences identical to those found in Asian elephants succumbing to fatal endotheliotropic herpesvirus infection.[61] Papillomatous lesions associated with herpesvirus also occur in Asian elephants.[54,55] Herpesvirus is discussed further in Chapter 11. Herpesvirus lesions are illustrated in Chapter 14.

## Mastitis

Botryomycosis (chronic connective tissue granulomas caused by *Staphylococcus* sp.) occurred in a captive African elephant and is the only reported case of mastitis the author is aware of. The left mammary gland was removed surgically. Interestingly, *S. agalactiae*, generally thought to be an obligate mammary pathogen, was isolated from a concurrent chronic tail folliculitis.[71]

## Tumors and Cysts

Cutaneous tumors are uncommon. Fibromas and fibrosarcomas have been reported in both African and Asian elephants and may occur in young elephants.[5,53,58] In one case, a 54-year-old elephant developed a rapidly growing mass proximal to a toenail at a site previously affected by pododermatitis. Metastases to the lung and axillary lymph nodes were found at necropsy.[41] Figure 18.8 (Color Section) depicts a fibrosarcoma in a 4-year-old Sumatran elephant observed by the author. Fibrosarcomas are generally slow growing, but surgical removal may be warranted if they are problematic or appear to be invasive. Tumors on the trunk may be particularly annoying for the elephant. Surgical removal of an aural rhabdomyoma from a wild-caught African elephant has been described.[42] Epidermoid cysts were incidental findings in random samples collected at necropsy from an Asian elephant[3] and a dermoid cyst was described in an African elephant.[62] Peribursal hematomas may occur with trauma, as reported in a working Asian elephant made to sit on rough roads. The condition may be very painful. Hematomas should be flushed and drained. A culture should be taken if infection is suspected and antibiotics (local or systemic) may be indicated.[63]

## Fungal Diseases

*Aspergillus niger, Candida albicans,* zygomycosis, *Trycophyton terrestre,* and *Microsporium* sp. have been recorded from foot and body lesions of captive elephants in Assam, India[10,12] (personal communication, Dr. Apurba Chakraborty, Assam, India, April 2005).

Mildly pruritic, widespread, hyperkeratotic skin lesions caused by *Trichothecium* sp., *Scopulariopsis* sp., and *Aspergillus* sp. were observed in two African elephants.[49] Figure 18.9 shows dermatomycosis in an Asian elephant,

***Figure 18.9.*** *Dermatomycosis in an elephant in Thailand (photo by Hank Hammatt).*

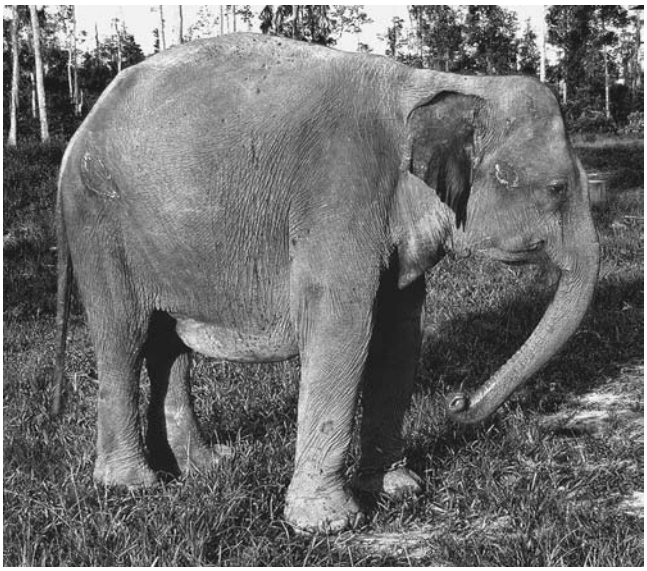

***Figure 18.10.*** *Ventral edema associated with recent trauma (photo by Hank Hammatt).*

thought to be caused by *Trichophyton sp*. A variety of saprophytic fungi may be isolated from lesions on the feet.[16] Topical antifungal agents are indicated. Extensive disease may require systemic therapy with griseofulvicin.

## Ventral Edema

Ventral edema ("dropsy" or "rot") may involve the ventral abdominal wall, the submandibular area, or the tissues surrounding the external genitalia. See Figure 18.10. A single, specific, underlying etiology has not been identified. Ventral edema has been associated with parasites,[8,15,83] kidney failure,[35,48] tuberculosis,[56,66] chronic diarrhea,[30] salmonellosis,[19] retained placenta,[50] and a wasting syndrome of unknown etiology.[20] Chronic and severe infection by *Fasciola jacksoni* invariably shows ventral edema (personal communication, Dr. Apurba Chakraborty, Assam, India, April 2005). Hypoproteinemia may be present, especially with severe parasitic infections. It is not always associated, however, and is probably not the sole underlying mechanism. Ventral edema occurs commonly among captive elephants in North America as the only clinical sign and has been variously treated with diet change, antibiotics, and diuretics. The majority of cases are non–life threatening, and resolve without treatment, with signs rarely persisting more then 3 months.[44] Ventral edema may be a nonspecific response to a variety of physiological stressors. Further research is needed.

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# 19 Musculoskeletal System

Gary West

## ANATOMY

Over 200 bones comprise the skeletal system of the elephant. There are some minor differences in skeletal anatomy between Asian (*Elephas*) and African (*Loxodonta*) elephants. *Elephas* typically has 19 pairs of ribs and 33 caudal vertebra, whereas, *Loxodonta* has 21 pairs of ribs and 26 caudal vertebra.[31,48,49] Also, *Loxodont*a typically have 4 toenails on the front foot and 3 on the hindfoot. *Elephas* typically has 5 nails on the front foot and 4 on the hindfoot. The anatomy of the foot is discussed in detail in Chapter 20. The ulna is larger than the radius, and these 2 bones are obliquely crossed. The carpus joint typically has 8 bones. Distal phalanges do not articulate with the middle phalanx on digits 2,3, and 4 on the front feet.[48] Epiphyseal cartilage is found in elephants throughout their lives.[48,49] See Figure 19.1 for a diagram of the overall skeletal structure of the elephant.

The most significant and vital muscular structures make up the trunk of the elephant. A complete and thorough description of normal elephant anatomy is beyond the scope of this chapter; the reader is referred to the additional references listed at the end of this chapter.

## EXAMINATION

Evaluation of the husbandry practices of an elephant herd is a critical part of the initial examination, and a comprehensive medical history is an important part of this initial assessment. A variety of questions regarding the herd's medical history and husbandry practices should be asked, including the following. What is the incidence of lameness in the herd? Is the herd in a breeding situation? How are the animals housed and restrained? What are the exhibit substrates? Do the animals participate in demonstrations or shows? Are there opportunities for appropriate amounts of exercise or digging behavior? What is the size of the exhibit? What are the animals being fed? What are the body weights of individuals? And what are the ages of the individual elephants?

Lameness is the most significant clinical sign associated with musculoskeletal disease in elephants. Evaluation of the animal's gait and movement may help recognize abnormalities. This evaluation should include observation of the animal's range of motion, stride, and its stance while resting. Asymmetry, swelling, or atrophy should be noted. Elephants with musculoskeletal disease may have difficulty getting up from a recumbent position, or they may be reluctant to lie down. Palpation of joints or muscles for heat, pain, or swelling should be included in the clinical examination.

### Imaging

Radiography is the most important imaging modality used in musculoskeletal evaluation of the elephant. Radiography is particularly useful for the detection of chronic changes in bones or joints of elephants.[13,17] Ultrasound has limited use in the evaluation of elephant musculoskeletal diseases but may be used for abscess detection and evaluation.[17] It also has application to joint disease, as discussed in Chapter 27. Magnetic resonance imaging or MRI is used for soft tissue evaluations in other species but this technology has not been comprehensively evaluated for large animal species. MRI may be useful for evaluating soft tissue injuries in the distal limbs of elephants. Thermography may be used to detect inflammation in tissues or "hot spots," especially after acute injury. Thermography may also be used to detect areas that lack nerve innervation or "cold spots." This has been used to evaluate trunk paralysis in captive elephants.[11] Thermography may be useful for initial evaluation of musculoskeletal diseases because of its ability to be used remotely. However, although thermography may localize a lesion, it cannot be used to establish a definite diagnosis. Computed tomography (CT) has not been used for distal limb evaluation in elephants except postmortem (see SECTION II in Chapter

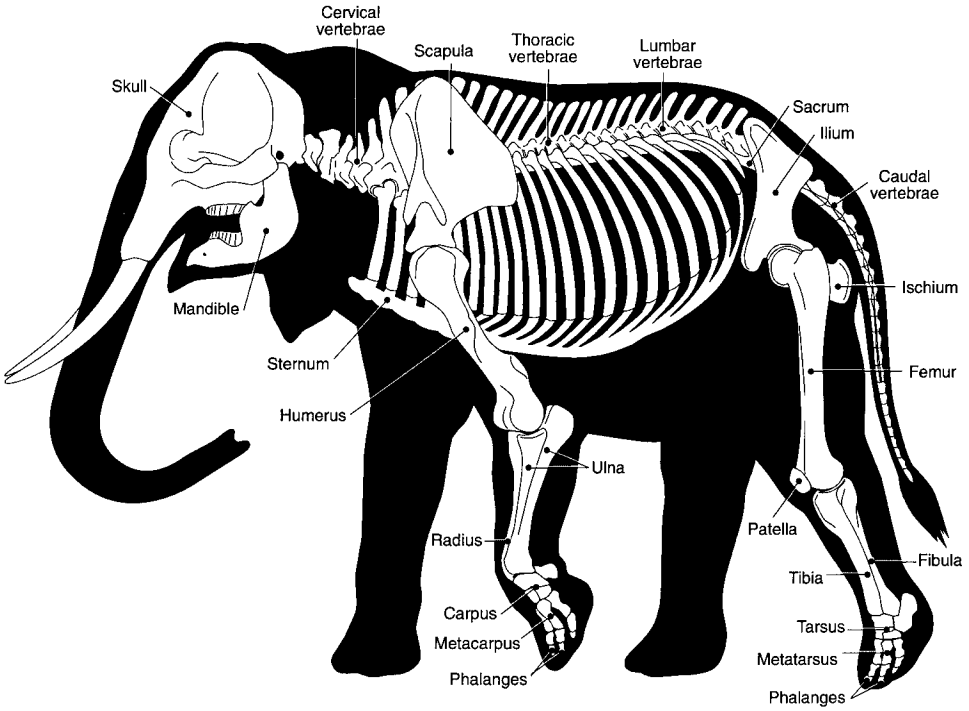


***Figure 19.1.*** *Skeleton of an elephant, drawn by D.J. Hillman, DVM (reproduced with permission of Indira Publishing House, West Bloomfield, Michigan).*

13). This imaging technology may be used, however, to further evaluate bony and cartilaginous changes. This could be helpful to detect more subtle causes of lameness in elephants. CT may not be practical because of the risk of general anesthesia and the large size of elephants. Nuclear scintigraphy is used in equine musculoskeletal examination. Scintigraphy could be performed on elephants and may be useful to diagnose causes of acute lameness, such as stress fractures. Stress fractures may be difficult to diagnose with conventional radiography and may be an underdiagnosed cause of elephant lameness.

## DISEASES OF THE MUSCULOSKELETAL SYSTEM

### Noninfectious Disorders

**Nutritional and congenital.** There have been several reported cases of fibrous osteodystrophy or nutritional secondary hyperparathyroidism in hand-raised elephant calves.[9,22] These elephant calves appear to grow and develop normally and then present with acute lameness or long-bone fractures. Elephant calves may present with chronic diarrhea prior to bony changes being detected, which may indicate they have a maldigestion or malabsorption syndrome.[9] Radiography reveals thinning of bone cortices and pathological fractures consistent with nutritional secondary hyperparathyroidism. See Figures 19.2 and 19.3.

At necropsy, cortical bone has been replaced with fibrous connective tissue and bones may often be cut easily with a sharp knife.

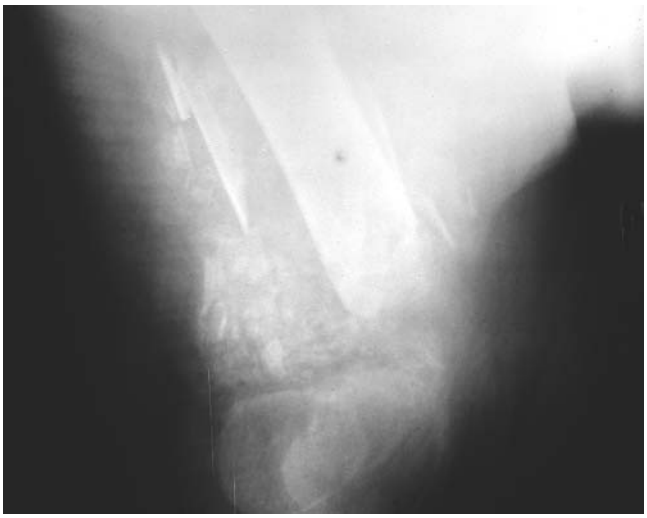

***Figure 19.2.*** *Radiograph of a bottle-fed Asian calf with metabolic bone disease. Note thinning of bone cortices and pathological fractures consistent with nutritional secondary hyperparathyroidism (courtesy of Dr. Charles Reid, Kennet Square, Pennsylvania).*

An African elephant with a tibiotarsal deformity was successfully treated with an orthotic brace.[45] This animal was thought to have nutritional secondary hyperparathyroidism previous to this deformity. Osteochondrosis may occur in elephants.[43] Abnormal cartilage formation and subsequent joint disease characterize this disease. Other factors—such as dietary influences, rapid growth, and heritability traits—may also influence the development of osteochondrosis. Nutrient and

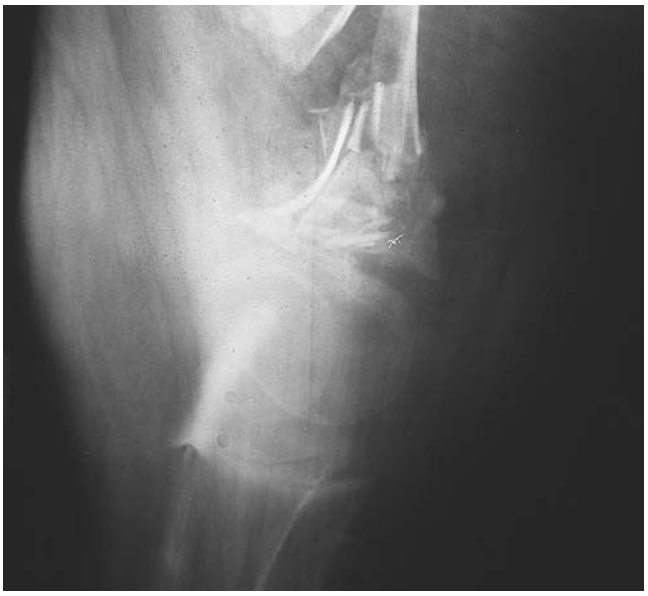

***Figure 19.3.*** *Radiograph from the same calf as Figure 19.2. Note multiple bone fragments.*

***Figure 19.4.*** *Fractures and dislocations are not necessarily fatal for elephants. In some cases, healing may be sufficient for survival and elephants may learn to compensate for their injury (photo courtesy of Carol Buckley, Hohenwald, Tennessee).*

mineral imbalances in elephant diets could contribute to abnormal cartilage formation.

Umbilical hernias have been reported in elephant calves.[1,53] These hernias result from a congenital defect in the muscle of the abdomen, resulting in herniation. Surgical intervention has been curative.[1]

Lameness due to high amounts of dietary protein was reported in a group of young African elephants.[50] Correcting the diet to include more fibrous material and lowering the protein intake seemed to be curative. This may reflect the occurrence of developmental orthopedic disease in young elephants, which could include osteochondrosis.

Vitamin E levels in elephant diets have been evaluated. Elephants more readily absorb specific forms of vitamin E, and diets should include acceptable levels of this form.[36] Hypovitaminosis E has not been diagnosed in elephants but low levels of this vitamin could contribute to the development of myopathy. See further discussion in Chapter 6.

**Trauma.** Sprains of the carpal joints are common injuries in logging elephants.[10] Traditional treatments include cold water hydrotherapy and gentle exercise after acute inflammation has subsided. Wounds resulting from chain restraint of elephants have been reported in captive elephants.[31,37] Wounds resulting from falling logs do occur in working elephants.[14] Also, trauma may be associated with chaining, tethering, and the use of an ankus in captive elephants.[14,31] Wild elephants may suffer gunshot wounds when engaged in human-elephant conflicts.[14,46]

Fractures in working elephants have typically carried a poor prognosis for return to work.[10] Captive elephants with fractures have been successfully treated.[21,29,33,38,41,51] Dislocations or luxations often carry a poor prognosis.[16] In some cases fractures or disclocations may heal on their own and elephants may learn to compensate. See Figure 19.4.

Traumatic diseases reported in elephants are often related to working accidents, performances, or loading for transport. Complete carpal luxation has occurred in circus elephants (personal communication, Dr. C. Reid, Kennet Square, Pennsylvania, 2005). In one case radiographs revealed a complete luxation of the proximal row of carpal bones from their articulation with the radius. The elephant was anesthetized and the luxation was manually corrected. A cast was applied to the affected limb. The elephant recovered, but eventually the joint reluxated and the elephant was euthanized. See Figures 19.5 and 19.6.

Therapy for traumatic injuries will depend on severity and location of the lesion. Rest is essential after acute injuries. Acute inflammation associated with these injuries can be treated with nonsteroidal antiinflammatories (NSAIDS). NSAIDS commonly used include flunixin meglumine, phenylbutazone, and ibuprofen. Studies on the pharmacokinetics of ketoprofen and ibuprofen in elephants have been done.[2,19] Wounds may be treated with topical disinfectants such as chlorhexidine solutions. In one case unpasteurized honey was used to treat a deep wound in an elephant.[47] Infected wounds may be treated with systemic antibiotics. Several pharmacokinetic studies of antibiotics in elephants have been completed, and therapy should be based on culture results or broad-spectrum antibiotics should be used.[8,25,28,34,39,42]

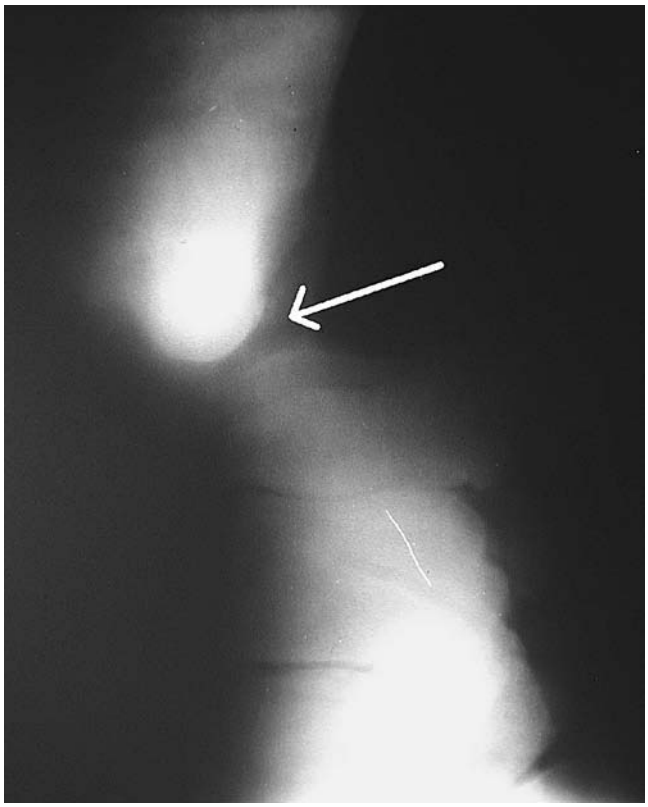

***Figure 19.5.*** *Radiograph of a 42-year-old Asian elephant with a complete dislocation of the carpal joint (courtesy of Dr. Charles Reid, Kennet Square, Pennsylvania).*

**Degenerative joint disease/osteoarthritis.** Degenerative joint disease (DJD) is one of the most common musculoskeletal diseases in captive elephants.[14,18,43,52] There is no single cause for the development of DJD. DJD may result from an imbalance of the integrity of the joint and the extrinsic forces placed on it. Typically, there are mechanical insults that contribute to the development of joint disease, but biological factors may also play a role. Mechanical trauma due to repetitive loading stress on hard surfaces is probably a major factor in the development of joint disease. Lack of sufficient exercise, excessive body weight, and poor conformation are other potential underlying factors. Conformation may concentrate stress and mechanical failure may result in the joint. Forgiving substrates that also allow opportunities for digging may help maintain joint health. Opportunities for swimming provide exercise for joints and allow animals to relieve some weight-bearing stresses. Traumatic events including hyperextension of joints may cause damage to joint margins, and this may lead to the development of joint disease. Occupational injuries can contribute to joint disease. Performance of certain behaviors may put excessive stresses on joints. Chaining elephants for prolonged periods limits their movement and may also contribute to the development of DJD. Animals that constantly pull or resist chaining may cause joint damage.

***Figure 19.6.*** *Cast applied to elephant carpal joint dislocation (courtesy of Dr. Charles Reid, Kennet Square, Pennsylvania).*

Also, reactive or infectious arthritis may result in the development of DJD. Reactive arthritis is discussed below.

Lameness is the most important clinical sign associated with joint disease. Unfortunately, lameness will be seen well after structural changes to the joint have occurred. Damage to cartilage results in the release of proteoglycans and other material into the synovial fluid. This will stimulate an inflammatory reaction and subsequent synovitis. Inflammatory mediators such as interleukins and prostaglandins further precipitate joint inflammation. These mediators will stimulate destructive enzyme release and joint damage results. Later in the development of DJD, a decreased range of motion in the joint may be noted. Decreased range of motion develops as a result of chronic inflammation and fibrosis of soft tissue structures. Advanced DJD may result in changes to the subchondral bone, which may lead to chronic bone pain. Bone pain may cause lameness even with antiinflammatory treatment.

Radiographic images of DJD may show specific signs depending on the joint involved and severity of the disease. Characteristic changes may include proliferative changes, lytic change, sclerosis, and decreased joint space. An important point is that radiographic changes are not always directly correlated to clinical signs and joint pathology. Additionally, radiographs may not be particularly helpful in identifying cartilaginous and soft tissue problems. Other imaging modalities, such as nuclear scintigraphy and magnetic resonance imaging, may be better to assess these structures.

Treatment for DJD should be a comprehensive approach and include a variety of treatments and also an evaluation of husbandry practices. Rest is essential after acute inflammation, and moderate exercise should begin after inflammation has subsided. Moderate exercise will not have a deleterious effect on the healing of cartilage. Swimming can be a good way for an animal to exercise while reducing weight bearing on the affected

joint. Elastic surfaces would also be important during exercise. Nonsteroidal antiinflammatory (NSAIDS) drugs are widely used to treat joint inflammation. These drugs interrupt the synthesis of prostaglandins. Prostaglandins are important mediators of inflammation and pain. Chronic use of NSAIDS may, however, suppress proteoglycan synthesis, which is an important constituent of cartilage. Therefore, NSAIDS are useful in acute inflammation but chronic use could contribute to cartilage loss. Corticosteroids are potent antiinflammatory drugs but can have detrimental effects. They would be contraindicated in reactive or infectious arthritis or in an elephant with unknown tuberculosis status. Also, corticosteroids may inhibit chrondrocyte development and the release of hyaluronan by the synovial membrane.

Hyaluronic acid or hyaluronan administration may benefit the soft tissue structures in joints and may improve lubrication. Hyaluronan may inhibit several inflammatory mediators. However, hyaluronan does not appear to benefit cartilage repair. Hyaluronic acid is available for intravenous administration in horses. The intravenous dose may be doubled and administered intramuscularly. Hyaluronic acid has been administered intramuscularly to elephants with joint disease with no deleterious effects. The benefit of this therapy has been difficult to measure.

Polysulfated glycosaminoglycan may have cartilage sparing and antiinflammatory properties. It is capable of inhibiting degradative enzymes and prostaglandins in articular tissues. This medication has been used in elephants with joint disease and appears to have some benefit. Doses are extrapolated from the equine dose, and it is administered intramuscularly. These compounds can be painful on injection. Oral supplements containing chondroitin and glucosamine are widely used in arthritic elephants due to the ease of administration. Efficacy and intestinal absorption of these compounds has not been proven, however. These compounds may increase proteoglycan synthesis and have antiinflammatory properties.

Exercise should be included in a treatment plan for elephants with musculoskeletal disease. Before extensive exercise is allowed, structural damage to soft tissue or bone should be ruled out. Access to large exhibits after an acute injury should be gradual because excessive activity could result in reinjury. During initial recovery periods exercise levels should be gradually increased.

Cold hydrotherapy has been used in elephants for a long time.[10] Hydrotherapy may reduce swelling and inflammation. An additional treatment modality that may have some efficacy is acupuncture. There are few reports of its use in elephants, but it may be an additional treatment modality.[55]

## Noninfectious Muscular Disease

Muscle cramping occurs in working elephants and may have severe consequences. Elephants that are hyperthermic after working may enter cold water and drown due to muscle cramping.[10] Vitamin E has been evaluated in elephants, and deficiencies of this vitamin may contribute to myopathy.[36]

## Infectious Diseases of the Elephant Musculoskeletal System

Infectious diseases of the musculoskeletal system include mycoplasmosis, tuberculosis, salmonellosis, myositis associated with *Clostridium septicum,* osteomyelitis, and septic arthritis. These diseases are discussed in detail in Chapters 6 and 11. Brief mention will be made here of tuberculosis (TB) and reactive arthritis.

**Tuberculosis.** In humans, tuberculosis (TB) may cause osteomyelitis, which may exhibit as foot ulcers or mimic pyogenic osteomyelitis.[54] Ten percent of extrapulmonary TB in humans results in chronic osteomyelitis.[54] In one case of an elephant with systemic atypical mycobacteriosis, the organism was isolated from the hip (personal communication, Dr. Kathyrn Gamble, Chicago, Illinois, June 2005). Tuberculosis should be considered in the differential diagnosis of musculoskeletal disease that has an unusual presentation or is nonresponsive to treatment.

**Joint disease.** A recent study of proboscidean skeletons confirmed the prevalence of joint disease in elephants.[40] This study found that joint disease in elephants resembled reactive arthritis or spondyloarthropathy. Reactive arthritis is characterized by immune complex binding and hypersensitivity to infectious agents. Elevated levels of rheumatoid factor, thought to be precipitated by a mycoplasma, have been found in arthritic Asian elephants.[6,7] *Mycoplasma* sp. were isolated from the urogenital tracts of 60% of the female Asian elephants in one study, and many of these elephants had arthritis.[6,7] An elephant-specific *mycoplasma* (*Mycoplasma elephantis*) was later isolated from the urogenital tract.[23] Mycoplasma has been cultured from an arthritic joint in a foal,[32] but there are no reports of isolation from elephant joints. Conclusive evidence that mycoplasma causes arthritis has not been established; however, further investigation may be warranted.

Acute inflammation and lameness have been observed in captive elephants with diarrhea. Enteric pathogens such as salmonella may elicit an immune response and subsequent reactive arthritis. Salmonella infections have been associated with severe diarrhea and stiffness in elephants.[5,20,44] Sexually transmitted diseases such as chlamydia (currently called *chlamydophila*) may also incite joint inflammation. Chlamydia has caused polyarthritis in a foal.[30] The foal and its dam did not have an antibody response and the organism was identified from the joint fluid, so investigating chlamydia as a cause for arthritis in elephants could be difficult.

If infectious agents are involved in the pathophysiol-

ogy of joint disease in elephants, antimicrobial treatment may be beneficial. A variety of antibiotics have been studied in elephants. Specific treatments should be directed to the primary pathogen involved. Mycoplasmosis may be treated with oxytetracyline or penicillins.[25,29,42] Enteric pathogens may be treated with amikacin, ceftiofur, or trimethoprim/sulfamethoxazole.[8,28,34] Nonsteroidal antiinflammatory therapy would also be beneficial during acute inflammation.[2,19]

The use of sulfasalazine may suppress inflammation and be useful in the treatment of reactive arthritis.[40] However, pharmacokinetic studies of sulfasalazine in elephants have not been done.

## Toxicities

Flaccid trunk paralysis in free-ranging African elephants may be caused by exposure or ingestion of toxins or toxic plants.[24] This disorder is discussed further in Chapters 30 and 33. Plants with cardiac glycosides have caused myocardial damage and death in African elephants.[3]

## Neoplasia

Neoplasia of the musculoskeletal system that have been reported include fibroma, fibrosarcomas, and a rhabdomyoma.[4,26,27,35]

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# 20 Foot Disorders

Murray E. Fowler

## INTRODUCTION

The prevalence of foot problems in elephants is unknown because no centralized reporting system has been established. In a retrospective study of 379 elephants, 50% were affected by foot disorders.[34] Few institutions have been spared the grief and frustration of dealing with elephant foot problems. It is the author's opinion that nonresolvable foot infection and arthritis are major causes necessitating euthanasia in elephants. It is unlikely that an elephant will progress through life without requiring periodic pedicures to keep the feet healthy. African elephants seem to have fewer foot problems than Asian elephants, but the reason for the difference is unknown.

A glossary of terms describing the elephant foot and foot conditions as seen by caretakers and veterinarians is found in Appendix 6.

Elephants' foot health would be enhanced if they lived in a natural habitat, which is defined as a large space with diverse topography and natural substrate. There should be wetlands, seeded and native pastures, wooded areas, natural year-round water (ponds, washes, streams, and dry creek beds), all with sufficient vegetation to maintain the elephants.[6] In a natural habitat, elephants spend their day walking, eating, bathing, and digging in soil. Natural vegetation, particularly browse, contains necessary trace minerals and vitamins that promote foot health. Elephants use their feet to help them forage. For instance, by placing a foot on a clump of grass, they pull grass blades between their toes which helps groom the interdigital skin and cuticles.[6]

It is not possible to provide the foregoing for captive elephants, except in rare situations. However, if elephant managers understand and appreciate the benefits of natural habitat to foot health, they might be better able to approximate natural habitat.

Facility design has an influence on foot health. Adequate drainage of floor surfaces in an elephant barn is critical. Architects often don't consider the amount of water necessary for cleaning and bathing or the volume of urine produced daily by elephants (54.4 L (5 gal)/4000 kg elephant). Drains are frequently installed that cannot cope with the volume of liquid or fail to capture debris before it enters the sewage system, causing obstructed sewage lines. See Chapter 5 for more detail.

Outdoor enclosure substrates are also important. Elephants should be allowed to dig. Clay soil, decomposed granite, or crushed limestone should be avoided because these tend to pack into pockets and crevices in the sole and behind the toenail. The best substrate is river-washed sand because it dries quickly in wet conditions.

## FOOT

The elephant foot is highly specialized to accommodate the heavy weight of the animal. The anatomy of the feet of Asian and African elephants is basically the same, with slight differences in shape and the number of toenails.[13,31,33,57,58] Basically there are five digits, which are not all identifiable externally, but some digits are represented by variable numbers of phalanges and toenails. Asian elephants usually have five toenails on each front foot (Fig. 20.1) and four on the rear foot (Fig. 20.2). African elephants generally have only four toenails on the front foot (Fig. 20.3) and three on the rear (Fig. 20.4). In general, only digits two, three, and four have three phalanges each (Fig. 20.5). Digit one may have only a metacarpal or also only one phalanx, and digit five has two phalanges. The distal phalanx (P3) may not articulate with P2. It is attached to a toenail by multiple laminae (Fig. 20.6).

The foot has an integumentary covering consisting of skin, toenails, and a cornified but flexible sole (slipper) similar to the bulb of the heel in a bovine (Fig. 20.7). See also Figures 20.1 to 20.4.

The bones of the feet have been well described by Smuts.[57,58] Elephant limb bones are massive and lack a

***Figure 20.1.*** *Right forefoot of an Asian elephant. Top: slipper; Bottom: lateral view.*

marrow cavity.[43,55] The marrow cavity is replaced with a network of dense cancellous bone, which provides hematopoiesis but renders the bones much stronger.[55] The bones of the front foot include the phalanges, metacarpal, and eight carpal bones arranged in two rows. The carpal bones are shortened and compressed and are an integral part of the foot (Fig. 20.8). The hindfoot is smaller than the forefoot and has an oval shape. The tarsus consists of seven bones arranged in three rows (Fig. 20.9).[58] Elephant toenails grow approximately 0.5 to 1.0 cm per month.

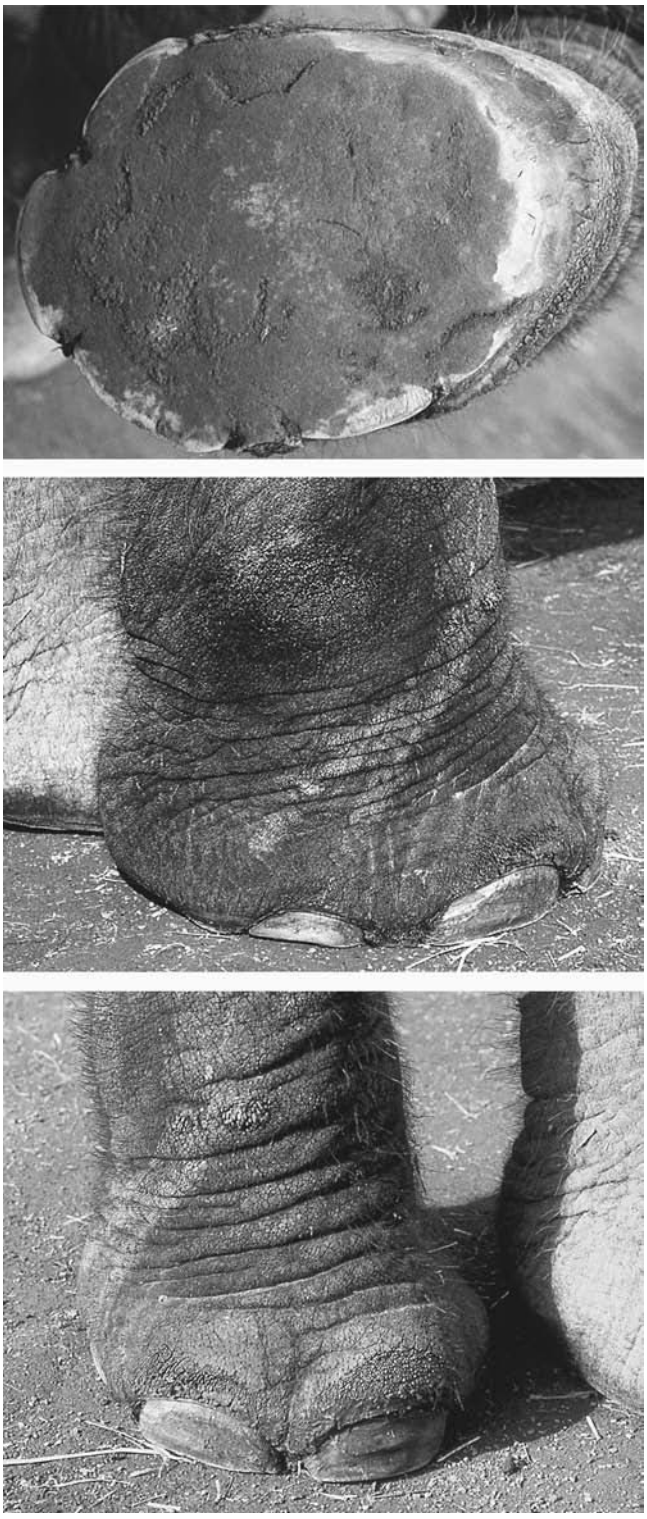

***Figure 20.2.*** *Right hindfoot of an Asian elephant. Top: slipper; Middle: lateral view; Bottom: front view.*

Each toenail has a cuticle similar to the human fingernail. Elephant toenails are not weight bearing as are those of a hoofed antelope. Two sesamoid bones lie on the distal palmar surface of metacarpals two to five. Metacarpal one has a single sesamoid bone. See Fig. 20.9.

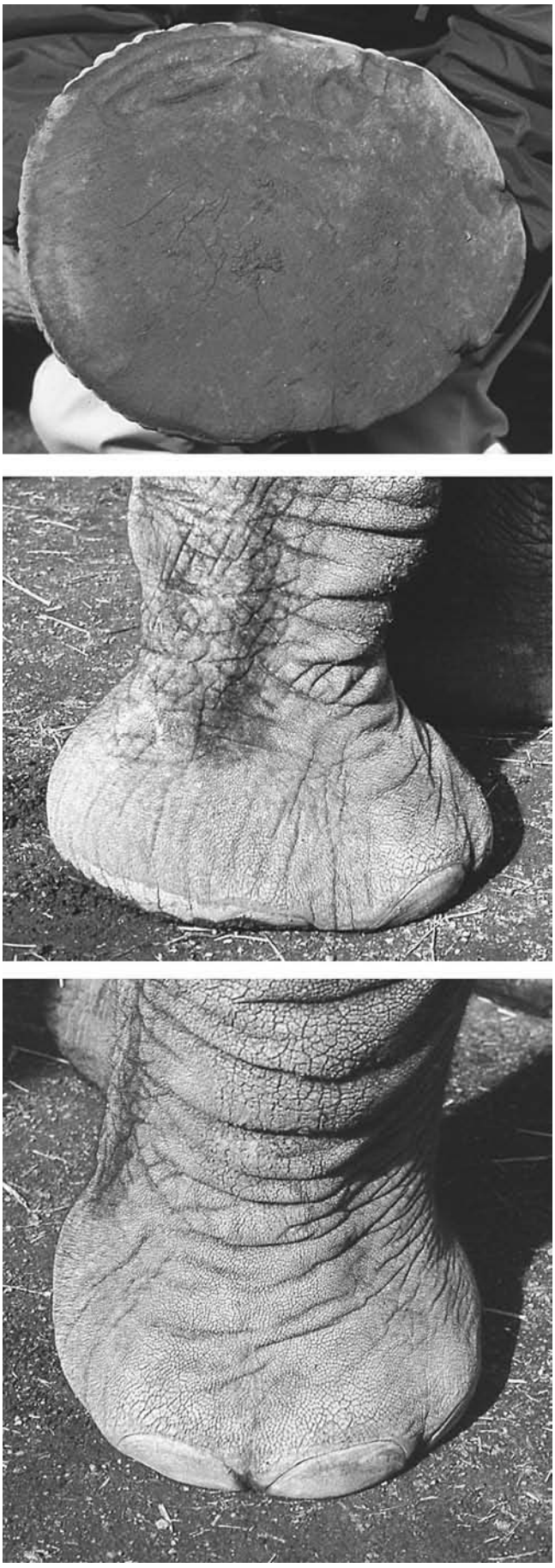

***Figure 20.3.*** *Right forefoot of an African elephant. Top: slipper; Middle: lateral view; Bottom: front view.*

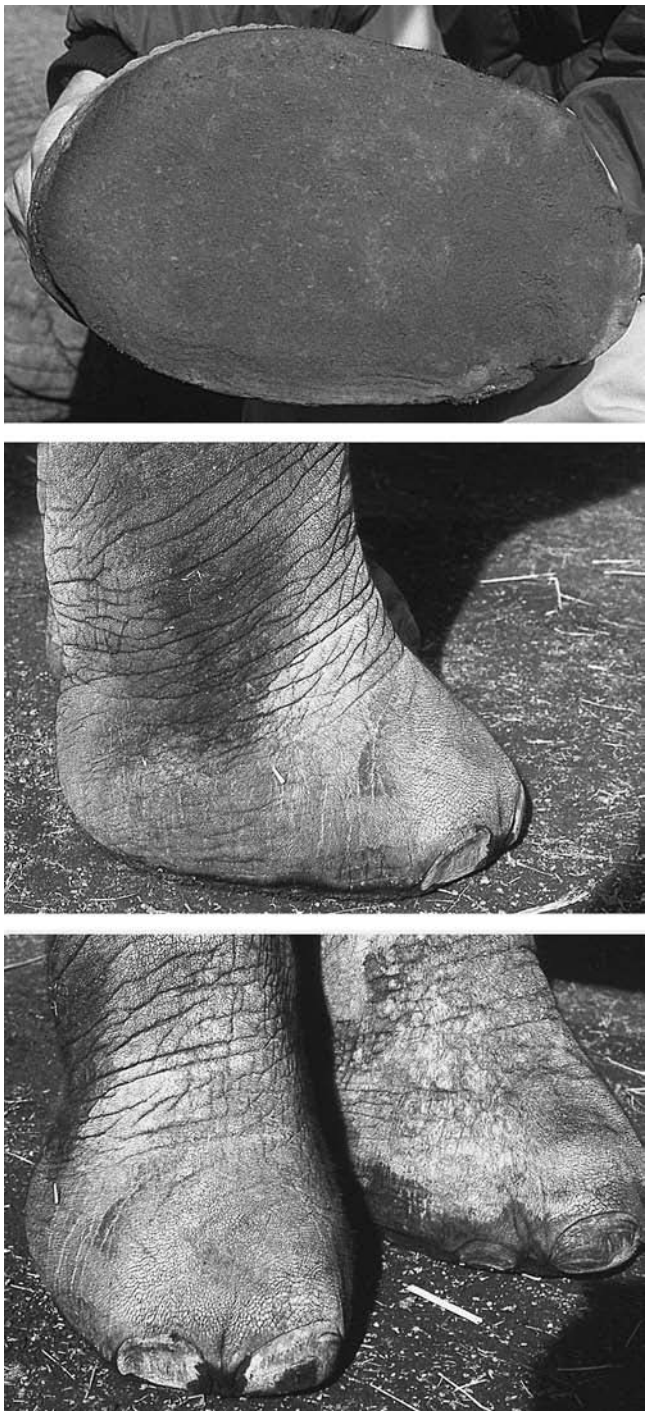

***Figure 20.4.*** *Right hindfoot of an African elephant. Top: slipper; Middle: lateral view; Bottom: front view.*

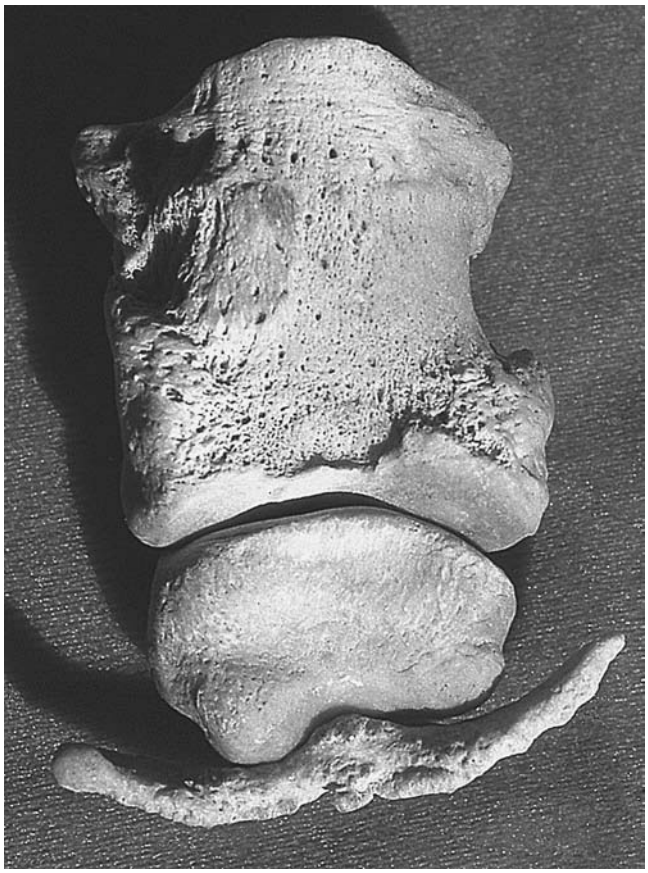

***Figure 20.5.*** *Phalanges of an elephant foot. Note winged P-3.*

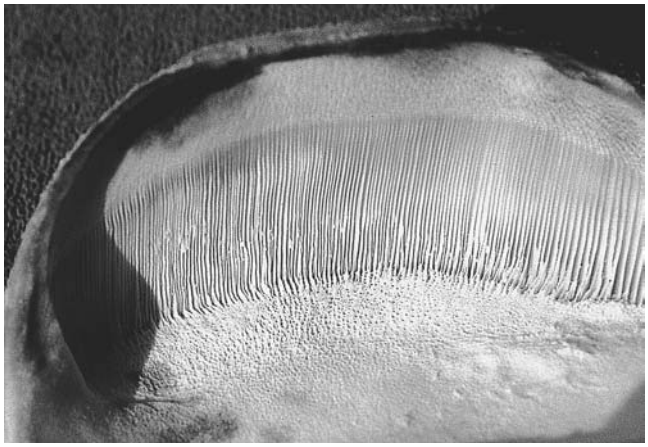

***Figure 20.6.*** *Laminae of an elephant toenail.*

The front foot has a unique cartilaginous structure (prepollex) attached to carpal bone one and metacarpal bone one by ligamentous tissue. It extends into the digital cushion and attaches to the sole slightly medial to the midline. Its function is presumed to be to stabilize the carpus and digits over the digital cushion. The hindfoot has a similar structure (prehallux) (Fig. 20.9).[30,58]

***Figure 20.7.*** *Sole of a juvenile elephant foot. Note the excessive sole that tends to entrap debris.*

Tendon and ligament arrangement in the foot is complex. Fascial sheets on the flexor surface of the metacarpals bind the digits together.[31,33,37] Flexion of the foot is provided by extensor and flexor tendons inserted on each digit as in other mammals. Lack of ability to flex the foot is an indication of either pain or ankylosis within the foot.

Elephants are semidigitigrade in the front feet, with the digits on the cranial and lateral aspects of the foot surrounding an extensive fibroelastic digital cushion (Figs. 20.10 and 20.11). The hindfoot is semiplantigrade. The metacarpal and metatarsal bones of the foot maintain a relative vertical angulation during weight bearing, but the phalanges compress the digital cushion and lie nearly horizontal when supporting the weight of the body.[33]

Muscles, tendons, collateral ligaments, synovial sheathes, vascular supply, and innervation are similar to those of other multidigit mammals.[30] Radiography of the ossification of developing bone was reported by Ayer.[3]

## PHYSIOLOGY OF THE FOOT

The foot of an elephant is a masterful piece of evolutionary development, designed to support the weight of the largest land mammal.[1,2] While standing, each foot of a large African male elephant (6000 kg) supports a weight of 1500 kg. That same elephant has an approximate slipper area of 1638.7 $cm^2$, which equates to a pressure of 0.92 $kg/cm^2$. While walking, with one foot swinging, the other feet support 2000 kg each for a pressure of 1.22 $kg/cm^2$. While ambling (modified pace), with only two

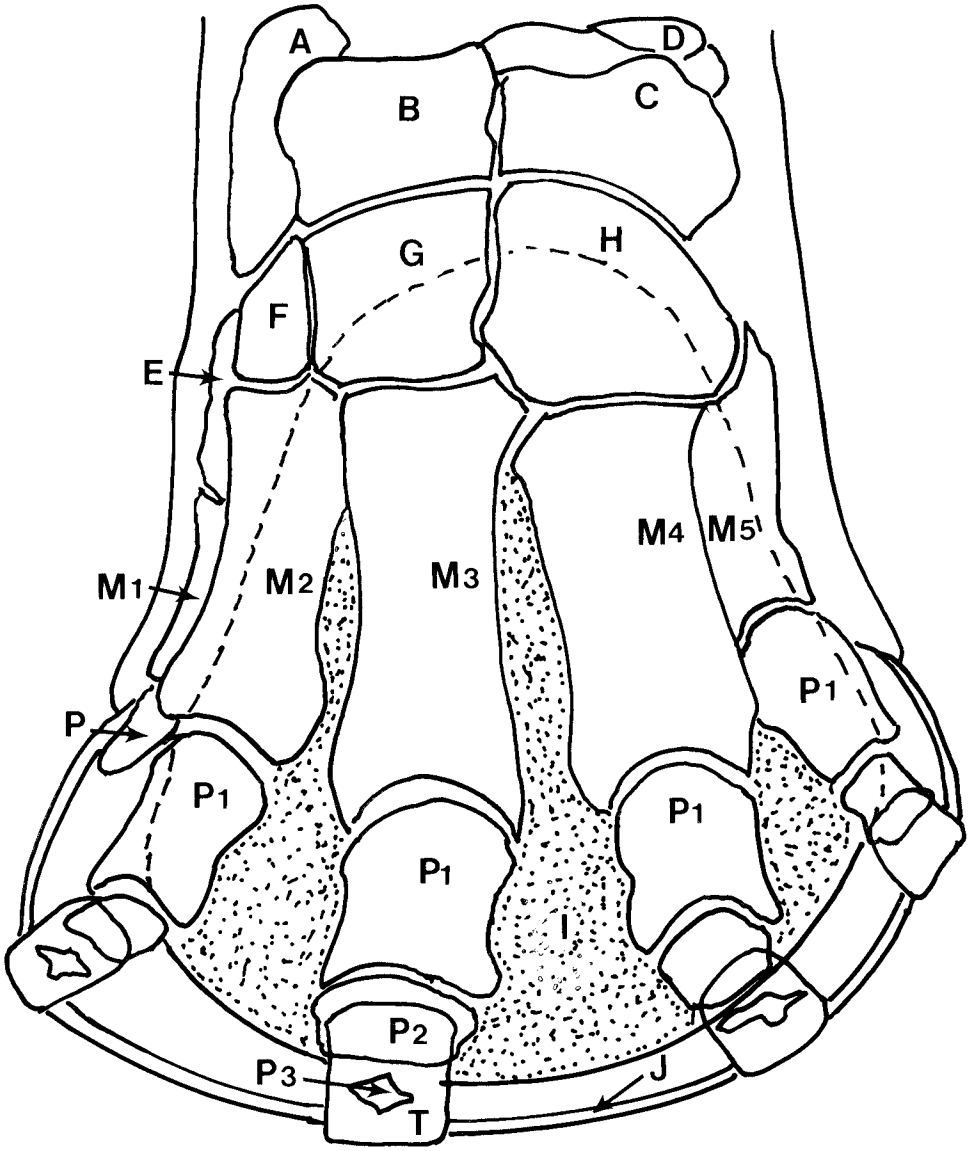


***Figure 20.8.*** *Diagram of a front view of the bones of an elephant forefoot. A) radial carpal; B) intermediate carpal; C) ulnar carpal; D) accessory carpal; E) carpal 1; F) carpal 2; G) carpal 3; H) carpal 4; I) digital cushion; J) sole (slipper), M 1–5 metacarpal 1–5, P 1–3 phalanx 1–3; S) sesamoid bone; T) toenail.*

feet supporting the body weight, each foot bears 3000 kg for a pressure of 1.83 kg/cm$^2$.

Both captive and free-ranging elephants (Asian and African), when standing and not otherwise engaged in some activity, move back and forth, alternating putting weight on one leg then the other. This is called *swaying,* and some people equate this only as stereotypic behavior. It is a natural behavior too and in the process of swaying back and forth the elephant is facilitating the circulation of blood from the distal extremities back to the heart.

Blood flows peripherally easily, but return flow must overcome the gravitational stagnation of blood in the long limbs. This is brought about by compressing the digital cushion in the foot, which acts as a peripheral pump to force blood up the leg. As the elephant alternates stepping on each foot, it facilitates circulation in the feet and legs.

In a small study conducted by the author on several elephants, weight bearing increased the circumference of the foot just above the nails from 5.0–11.4 cm or by 7.0–9.7%. As weight is applied, the digital cushion compresses and pushes peripherally, causing the increase in circumference and at the same time compressing the veins in the foot.

## ROUTINE FOOT CARE

More caretaker time is spent caring for captive elephant feet than in any other task, except feeding and cleaning. Constant attention must be given to ensure that all structures of the foot remain healthy.[10,15,16,50,51,64,65] In either free contact or volunteer contact behind a barrier, an elephant should be trained to lift each foot for inspection and place it on a pedestal, tub, or bar.[27,28] The bulk of an elephant precludes a person from manually

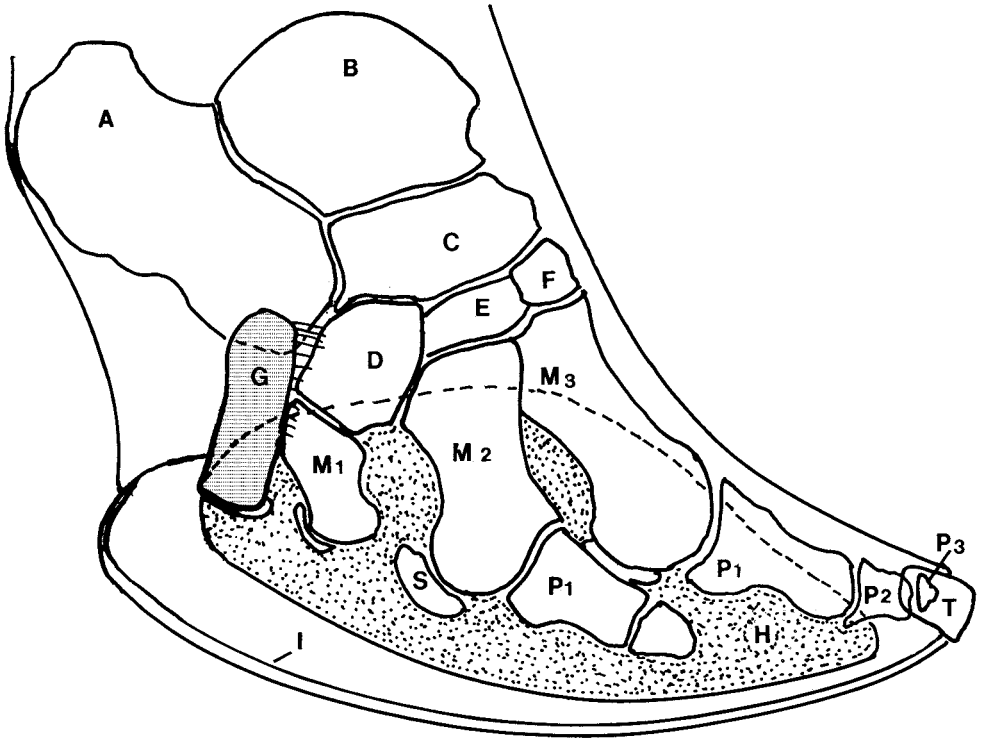


***Figure 20.9.*** *Diagram of a lateral view of the bones of an elephant hindfoot. A) calcaneus; B) talus; C) central tarsal; D) tarsal 1; E) tarsal 2; F) tarsal 3; G) prehallux, M 1,2,3 metatarsal 1,2,3, P 1,2,3 phalanx 1,2,3; H) digital cushion; I) sole (slipper); S) sesamoid bone; T) toenail.*

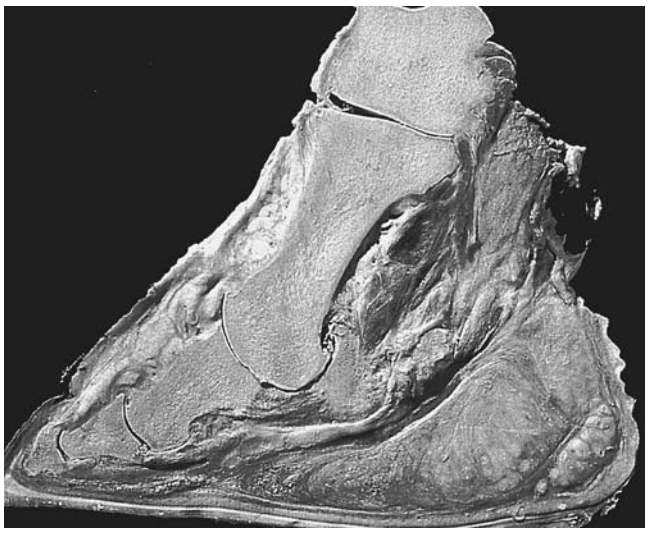

***Figure 20.10.*** *Sagittal section of an elephant forefoot.*

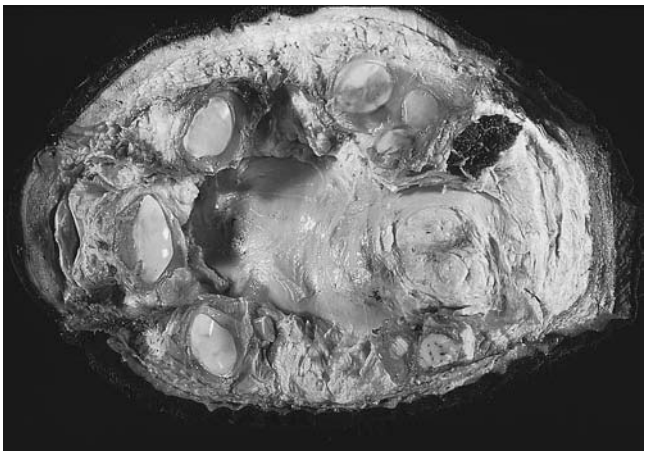

***Figure 20.11.*** *Dorsal view of the digital cushion (A), phalanges (B), and associated tissues.*

lifting the leg and holding it in position against the will of the elephant, unless a block and tackle or winch is used. Such force is not recommended.

## Components of a Foot Care Program

Components of a good foot care program include experienced staff, trained elephants, appropriate tools and equipment, proper facility design, and sufficient time for handlers to provide the necessary care.[14,24,45,46]

All elephants should be vaccinated with tetanus toxoid. In any puncture wound or cases of severe pododermatitis, a booster dose of toxoid should be administered. If the elephant has not been vaccinated previously, the author would nevertheless recommend that tetanus toxoid be administered, not tetanus antitoxin. Antitoxin is prepared from horse serum, and a risk exists for anaphylactic shock should the elephant be sensitive. Anaphylactic shock is discussed in Chapter 15.

Asian elephants are more likely to develop foot problems than Africans, the reasons for which are not known.

Perhaps evolutionary habitat may have had some bearing. For instance, African elephants rarely have toenail and cuticle problems because they evolved in open savannahs that required walking as much as 18 hours a day to obtain forage and water. The sole developed characteristics that allowed constant wearing. However, sole overgrowth must be dealt with in captive populations.

Asian elephants on the other hand evolved in moist climates with lush forage. They have less problems with sole overgrowth, but more challenges with toenails and cuticles.[46]

Any treatment regimen that inflicts pain must include an analgesic; otherwise, the elephant may resist any future attempt to handle its foot.

The feet should be inspected and cleaned daily. The bottom of the foot should be brushed with a stiff-bristled brush and checked for foreign bodies imbedded in any of the cracks or grooves of the slipper or behind the toenail. Objects may be removed with either a hoof knife or hoof pick as would be used on a horse's foot. Clean pockets associated with an overgrown slipper of fecal material or accumulated debris. Clean between the toenails and at the margin of the skin at nails and slipper and check for swelling, excessive heat, exudate, or fetid odor. If an elephant is allowed to stand in mud, water, or its own excrement for an extended period, the skin may become macerated and secondarily infected.

Check the nails, cuticle, and slipper for overgrowth and schedule appropriate trimming. It may be necessary to immobilize untrained elephants to accomplish even simple tasks, such as examining the foot or cleaning out debris. Sedation or tranquilization only encourages the elephant to resist lifting a foot. However, a tilting elephant restraint device has been used effectively without anesthesia.[52]

Radiography of the foot is discussed elsewhere in the book but it should be mentioned here that reference radiographs should be on file in case of a problem later.

## DISEASES

Foot problems constitute the single most important ailment of captive elephants.[15,16,35] Elephants in work camps in India and Southeast Asia suffer from problems similar to those of captive North American Asian elephants.[7,42,54] In one elephant camp, it was estimated that 50% of the elephants suffered from one or more foot problems (personal communication, Professor D. K. Lahiri-Choudhury, Portland, Oregon, March 20, 1998). The true prevalence is unknown because of the difficulty in collecting data.[21]

One often hears the statement that foot problems don't occur in free-ranging elephants. That is not true, because wild elephants may suffer from snare injuries, lacerations, fractures, penetration of foreign bodies, and injuries caused by stepping on land mines. Pododermatitis occurred in several wild African elephants in Kruger National Park in South Africa.[29]

Diseases will be discussed according to the organ system involved.

### Integument

**Skin.** The skin may be subjected to lacerations, foreign body penetration, contusions, abrasions, burns, and maceration. The latter is a major management concern necessitating a sound sanitation program. Prolonged irritation of the skin of the foot may lead to hyperkeratosis, particularly at the junction of the skin and sole. Such hyperkeratosis may be a prelude to cracks of the epidermis. The skin should be inspected periodically, particularly during wet weather or winter to recognize and alleviate cracking and avoid secondary infection (pododermatitis).

Hyperkeratotic skin may be thinned by gentle abrasion using coarse-gained sandpaper or a hoof rasp. The skin may be softened with a mixture (50:50) of vegetable and mineral oil. Ointments tend to accumulate dirt and debris.

#### Infectious diseases of the skin of feet.

Foot and mouth disease (FMD). FMD is a rare disease of captive elephants.[26] The details are discussed in Chapter 11. The clinical signs seen with foot involvement are as follows: The first signs noted are anorexia and lameness. The skin around the toenails and margins of the slipper may be hot, swollen, and tender. Lameness may be so severe as to cause the elephant to remain recumbent. The slipper may become undermined resulting in sloughing. Nursing care may be required for months.

Elephant pox. Elephant pox has been reported in Europe. The toenail and slipper corium may become infected, necrotic, and odoriferous. The toenails of severely affected elephants may slough.[22] Details of elephant pox and references may be found in Chapter 11.

Pododermatitis. The term *pododermatitis* is used to describe any infectious process of the foot, which may be as simple as a localized abscess or as complex as a generalized infection in and around the nails or in pockets within and beneath the sole (Fig. 20.12). A severe infection may spread to involve the bones and joints of the foot, producing septic osteitis and arthritis. A serious consequence is infection of the digital cushion.

Predisposing factors include neglect of regular nail and sole trimming, constant exposure to filth and moisture and lack of routine inspection of the undersurface of the foot. Sedentary elephants are more likely to develop foot infections than active elephants. Elephants with conformational faults tend to develop foot problems as they begin to age (30–40 years) because they walk in such a manner that unequal weight is distributed to unaffected limbs.[20]

***Figure 20.12.*** *Pododermatitis.*

An outbreak of pododermatitis occurred in free-ranging African elephants in Kruger National Park.[29] The outbreak was restricted to specific locations of harsh shrubby vegetation (mopane scrub, *Calophospermum mopane*). A prolonged drought and overbrowsing left considerable mopane stubble that apparently penetrated the slippers of the forefeet of heavy bulls (60–65% of the weight is borne on the forequarters). The elephants congregated around provided water tanks and splashed water out of the tanks resulting in mud mixed with urine and feces, providing the right milieu for infection. Signs included lameness, visible flaps of sole, and sloughing of the sole. Regional (axillary) lymph nodes became involved with swelling and drainage. Organisms isolated from the lymph nodes were the same as those isolated from the feet.[29]

Infectious agents vary from locality to locality. No studies have been conducted to ascertain the normal microflora of elephant feet. Numerous microorganisms are associated with lesions of the feet. Most of them are contaminants or opportunistic pathogens found in unsanitary environments contaminated with feces and urine. Multiple organisms may be isolated from the same lesion.

Common isolates are enteric bacteria (Enterobacteriaceae), which are gram-negative, non–spore-forming rods. They usually require an enriched medium for successful culture. They may be isolated from feces, pastures, and bedding and from the digestive tract of healthy elephants. Enteric genera may be pathogenic, opportunistic, or simply contaminants.

Enteric genera that have been isolated from foot lesions in elephants include Aeromonas, Citrobacter, Escherickia, Klebsiella, Morganella, Salmonella, and Proteus.[8,29]

*Streptomyces keratolytica*, a fungus, was associated with parasitic lesions of the feet of Asian elephants (62.5%). Other fungal organisms isolated from superficial lesions or observed in tissue sections included *Fusarium solani, Penicillium vermiculatum, Penicillium allulacecum, Paecilomyces lalanicnus, Emericellopsis synnematicolor, Cladisporum oxysporum,* and *Cryptococcus neoformans*.[8] A yeast *Candida albicans* has also been isolated from foot lesions.[5] Tables 20.1 and 20.2 list other organisms isolated from elephant foot lesions.

Clinical signs of pododermatitis include lameness, obvious overgrowth of the keratinized structures, fetid odor, and exudation from around the toenails. Rubber gloves should be worn when examining and treating suspected pododermatitis, because the odor is pervasive and persistent.

Adequate drainage of all pockets, tracts, and grooves is the key to management of pododermatitis (Figs. 20.13, 20.14, and 20.15). Foot soaking with a disinfectant solution or antibiotic therapy is superfluous if adequate drainage is neglected. The foot should be thoroughly cleaned and manicured. Trimming a severely infected foot is laborious and time consuming, and usually necessitates immobilization. Infections are common beneath and surrounding the toenails. If the elephant has not been on a routine tetanus toxoid vaccination regimen, this should be instituted immediately, because anaerobic pockets are a natural nidus for *Clostridium tetani*.

Infections involving the digital cushion are extremely difficult to treat. This tissue is relatively avascular, which slows the healing process. It is also difficult to establish drainage, because the surrounding elastic tissue expands to block a drainage window.

Special boots or sandals have been constructed to protect the dermis, keep topical medication on a lesion, provide ventilation, and deflect solid contaminants to keep the lesion reasonably clean (Fig. 20.16).[25,66] Such devices must be accepted by the elephant and involve intensive care on the part of handlers. Elephants are not likely to leave a bandage on their feet without constant supervision by a handler.

Soaking an elephant's foot in a disinfectant solution is a time-honored practice. No single solution is universally accepted by elephant veterinarians (Table 20.3). The author has used chlorhexidine, povidone iodine, Epsom salt, and copper sulfate solutions. A single, ideal regimen cannot be recommended, but it is wise to avoid concentrating the solution more than recommended by the manufacturer, because some of these solutions may be irritating to exposed tissue.

The use of local or parenteral antibiotic therapy is a matter of choice. Generally the author would not recommend antibiotics unless the bone, tendon sheath, or a joint is involved. Selection of an antibiotic should be based on culture and sensitivity.[53] A recent technique for regional digital intravenous antibiotic perfusion based on equine therapy has been employed in elephants.[60,61] Infection may begin behind the nail and migrate up the laminae to the cuticle area (gravel in a horse). A hot, tender, swollen area may be noted at the

**Table 20.1.** Characteristics of Organisms Isolated from Elephant Feet

| Organism | Gram Stain/Shape | Oxygen Requirements | Spore Status | Natural Habitat | Toxins |
|---|---|---|---|---|---|
| *Streptococcus agalactiae** (1,4) | + / cocci | Aerobe to facultative anaerobe | No spores | Commensal in upper airways and G.I. tract | None |
| Beta hemolytic streptococci,* (1,2,3,4) | + / cocci | Aerobe to facultative anaerobe | No spores | Commensal in upper airways and G.I. tract | None |
| *Staphylococcus aureus*, (1,2,3,4) | + / cocci | Aerobe to facultative anaerobe | No spores | Mucocutaneous borders, transient in G.I. tract | Exotoxins |
| *Prevotela melanogenica*, (4) (Peptostreptococcus) | + / cocci | Obligate anaerobe | No spores | Normal flora | Exotoxin |
| *Corynebacterium* spp., (1,4) | + / coccoid rod | Aerobe to facultative anaerobe | No spores | Normal inhabitant of oral cavity and G.I. tract | Exotoxin |
| *Clostridium tetani,** (3,4) | + / rod | Obligate anaerobe | Terminal spore former | Feces, soil, necrotic wounds | Exotoxin |
| *Bacillus cereus* (4) | + / rod, related to *B. anthracis* | Facultative anaerobe | Spore former | Soil, contaminated food; causes food poisoning in humans | |
| *Eggerthella lenta* (4) *(Actinobacterium*, Eubacterium) | + / Diphtheroid, related to *Corynebacterium* spp. | Obligate anaerobe | No spores | G.I. tract | Exotoxin |
| *Pseudomonas aeruginosa** (1,3,4) | - / rod | Obligate aerobe | No spores | Soil, water, transient in feces of normal animals | Exotoxin |
| *Aeromonas hydrophila* (3,4) | - / rod | Aerobe to facultative anaerobe | No spores | Soil, water | |
| *Pasteurella multicida, P. haemolytica* (4) | - / coccobacilli | Aerobe | No spores | Mucous membranes of oropharyngeal region | Exotoxin |
| *Mannheimia haemolytica,* (4) (*Pasteurella*) | - / coccobacilli | Aerobe | No spores | Mucous membranes of oropharyngeal region | Exotoxin |
| *Dichelobacter nodosus** (Fusiformis, Bacteroides) (4) | - / rod | Obligate anaerobe | No spores | Normal flora of skin; the infectious agent of ovine foot rot | Exotoxins |

*= Important pathogen; G.I. = gastrointestinal; 1 = Boardman 2001; 2 = Chatterjee 1984; 3 = Gage 1997; 4 = Keet 1997.

top of the nail. The swelling may rupture, and an odoriferous exudate is discharged.

**Toenails.** Elephant toenails require constant attention from caretakers to prevent hidden infection from progressing to untreatable osteomyelitis.

Overgrowth. Toenails grow primarily at the germinal epithelium at the root (top) of the nail, at a rate of approximately 0.5 to 1.0 cm per month. If the enclosure substrate is not abrasive or the elephant does not walk as much as it should, it may be necessary to remove excess nail every 2–3 months. Trimming is performed using equine hoof nippers and a hoof rasp (Fig. 20.17).

Neglected toenails may become infected (Fig. 20.18), become deformed, or grow laterally and penetrate into adjacent skin or sole. This causes an inflammatory response (perionychia) similar to that caused by a human ingrown toenail. Signs include lameness, evidence of the embedded toenail, sensitivity to palpation, exudation, and formation of granulation tissue. Elephant granulation tissue presents as a whitish, friable mass.

Toenail cracks. Horizontal and vertical cracks may occur, but vertical cracks are more common (Fig. 20.19).[38,40,47] Cracks may begin at the cuticle and extend distally to the tip, or they may begin at the bottom and extend proximally. Cracks may be superficial, being confined to the keratinized nail, or extend into the corium, which causes more discomfort for the elephant.

The etiology of toenail cracks is unknown, but it may include factors such as nutrition, genetics, overgrowth, and trauma. When a crack develops, it is exacerbated by the expansion and contraction of the foot during ambulation.

Treatment of superficial cracks may require little more than grooving with a hoof knife to determine the depth of the crack. Avoid rasping the outer surface of the nail with superficial cracks, because this removes the protective periople of the nail. Cracks into the corium necessitate more aggressive treatment. Groove the crack with a sharp hoof knife or groover until all vestiges of the crack (black tracts) are opened. As the corium is approached, a gauze sponge soaked with 2% lidocaine should be applied to the crack and left in place momen-

**Table 20.2.** Microorganisms Isolated from Foot Infections and Abscesses in Elephants*

| | Gram Stain | Shape | | Oxygen Requirements | | | Motility | | | Pathogenicity | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Microorganism | Neg. | Rod | Coccus | Aerob. | Anaer. | Facult. | Anaer. | Motile | Nonmotile | Primary | Opport. | Comment |
| *Escherichia coli* | | X | X | | X | | | X | | X | X | Zoonosis |
| *Proteus vulgaris* | | X | X | | | | X | X | | | X | Cultures overgrow other organisms |
| *Pseudomonas aeruginosa* | | X | X | | | X | | X | | | X | Zoonosis |
| *Fusobacterium necrophorum* | | X | X | | | X | | | X | | X | |
| *Dichelobacter (Bacteroides) fragilis* | X | | X | | | X | | | X | X | X | Zoonosis |
| *Dichelobacter nodosus* | X | | X | | | X | | | X | X | X | Also isolated from regional lymph nodes |
| *Beta hemolytic streptococci* | X | | | X | X | | | | X | X | X | Zoonosis |
| *Streptococcus agalactiae* | | X | | X | X | | | | X | X? | X? | |
| *Staphylococcus aureus* | X | | | X | | | X | | X | X | X | Grow in clusters; zoonosis |
| *Pasteurella multocida* | | X | X | | | | X | | | X | X | Bipolar staining |
| *Aeromonas hydrophila* | | X | X | | | | X | X | | | X | |
| *Enterococcus zymogenes* | X | | | X | | | X | X | X | | X | |
| *Salmonella* spp. | | X | X | | X | | | | X | X | X | |
| *Klebsiella* spp. | | X | X | | | X | | | X | X | X | Zoonosis |

*Holt, J.G., Krieg, N.R., Sneath, P.H.A., Staley, J.T. and Williams, S.T.1994. Bergey's Manuel of Determinative Bacteriology, 9th ed. Baltimore, Williams and Wilkins.

***Figure 20.13.*** *Sole of an adult elephant's foot with excessive growth, producing grooves, ridges and pockets.*

***Figure 20.14.*** *Trimming the sole with a drawing knife.*

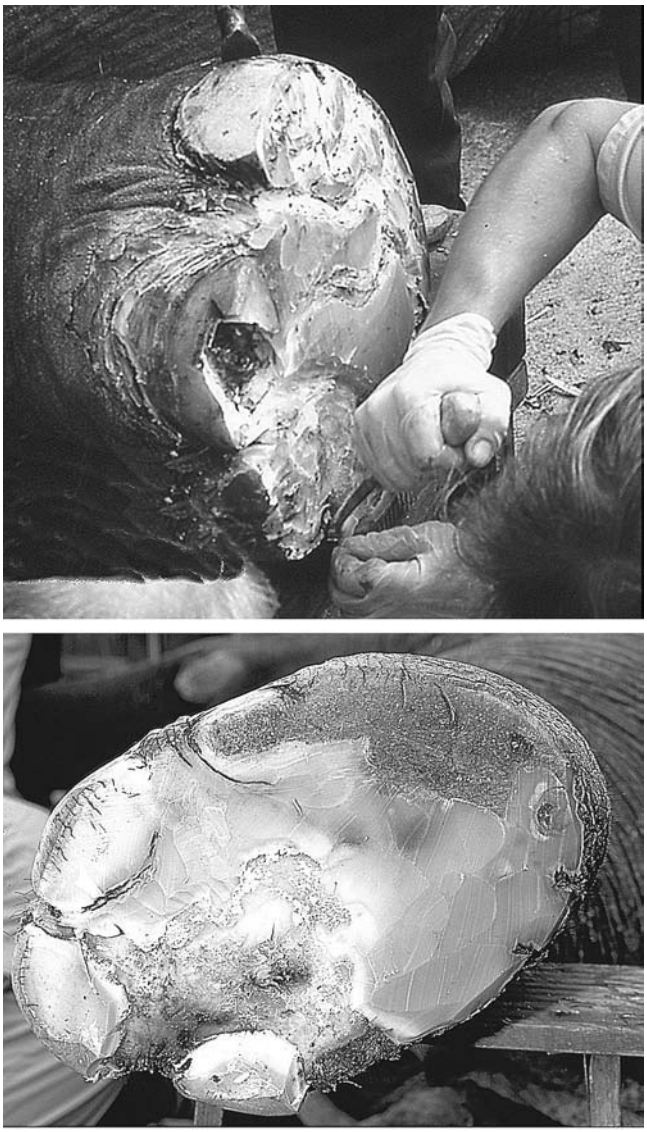

***Figure 20.15.*** *Trimming a foot. Top) Using a hoof knife; Bottom) extensively trimmed.*

tarily to provide local anesthesia. Infliction of pain should be avoided or it is difficult to proceed.

Deep cracks require thinning of the edges to alleviate the mechanical pressures that tend to force the crack open. This is done by rasping the surface of the nail to feather the crack edges to the corium. At the same time, the distal end of the nail should be shortened as far as possible to prevent ground pressure. The corners of the nail should be rounded. If the crack has not reached the cuticle, a horizontal groove at the top of the crack may discourage further cracking.

The author has had no success with clamps, clips, staples, screws, or tension wires, as used in horses. None of these can withstand the tremendous pressures exerted on the toenail by the expansion of the elephant's foot. A protocol using epoxy resin and fiberglass fabric bonded to the nail with vertical grooves produced by a hobby drill (Dremel Moto-tool) has been reported. This is a procedure similar to one performed routinely on the fractured carapace of chelonians.

***Figure 20.16.*** *Sandal on an elephant's foot.*

In all cases, the healing process is prolonged and interrupted with periodic exacerbations.

Onychia. Inflammation or infection of the toenail bed is usually the result of failure to clean behind the nail regularly. Anaerobic conditions are set up, and infection spreads in the path of least resistance, which is upward through the laminae to rupture at the top of the nail (equine gravel). Clinical signs are lameness; a hot, painful swelling; or a draining tract at the top of the nail. Examination of the bottom of the nail reveals a black tract extending dorsally. A severe onychia may cause separation of the nail from the corium with a subsequent slough.

A differential diagnosis must consider the fluid pockets that occur at the base of toenails beneath the cuticle. Such pockets are filled with sweat, not pus.

Treatment involves trimming out the black tract and packing it with a disinfectant-saturated gauze pad to prevent debris from being repacked into the void, Table 20.4. The pack should be changed at least twice daily. It is also recommended that a sandal be affixed to the foot.

Cuticle. The cuticle is the keratinized skin at the junction with the nail. Elephant cuticles should be manicured regularly. Some elephants' cuticles seem susceptible to excessive growth (hangnails), but inflammation causes proliferation. Thus animals kept standing in mud or their own excrement are most likely to develop problems. The keratinized cuticle becomes hardened and eventually cracks, causing formation of painful hang-

**Table 20.3.** Solutions Used to Soak Elephant Feet

| Generic Name | Trade Name | Source | Indications | Mixing Directions | Comments |
|---|---|---|---|---|---|
| Magnesium sulfate, USP, $Mg\ SO_4\ H_2O$ | Epsom salt | Any drugstore or pharmacy | A concentrated solution of Epsom salt is hypertonic and draws fluid from tissue; used for local inflammation, cellulitis, arthritis, and contusions | For an elephant foot, 225 g (0.5 lb) of Epsom salt in 2 l (2 quarts) hot water; allow water to cool | |
| Chlorhexidine diacetate | Nolvasan solution, 2% chlorhexidine | Fort Dodge Laboratories, 800 5th St., N.W. (P.O.B. 717), Fort Dodge, IA 50501 | General disinfectant | 250 ml (9 oz) of the 2% stock solution to 1.0 l (1 quart) of clean water = 0.5% chlorhexidine | Not effective against *Pseudomonas* spp., or gram-positive cocci |
| Povidone-iodine solution; other names include iodophore, tamed iodine | Vedadine, 10% stock solution | Vedco, St. Joseph, Missouri, USA | General disinfectant | May use undiluted or for irrigation or soaking; dilute stock solution 1:10 (400 ml to 3.79 l (1 gal) | May dilute up to 1:100 |
| Copper sulfate | Copper sulfate | Veterinary supply companies | Disinfectant | 50 g to 1 liter = 5% solution | Caustic in high concentrations |
| Sodium hypochlorite (NaOCl) | Clorox, 6% solution | Any grocery store | Powerful oxidizing agent and disinfectant | Use 0.25% solution for soaking; 155 ml bleach to 3.7 l (1 gal) water | Will bleach clothing |

Formula for calculating dilution of a stock solution to a therapeutic solution:
the % of the active ingredient in the stock solution $\times X$ = the desired ultimate % $\times$ the volume desired.
$5\% \times X = 0.25\% \times 1000$ ml
$X = 250/5 = 50$ ml of 5% stock solution in 750 ml of water

***Figure 20.17.*** *Rasping a toenail.*

***Figure 20.18.*** *Chronically infected toenail.*

nails (Fig. 20.20). Furthermore, the crack is a portal for infection.

A condition that may develop in some elephants is an extension of an overgrowth of the cuticle, which appears as a thickening of the internail skin. Sometimes it is described as an interdigital callus, similar to that seen in the bovine foot. The interdigital skin should be flexible and free of infected pockets. If this skin becomes heavily keratinized and hardened, it acts as a foreign body and causes discomfort when walking by pinching the skin between contiguous nails.

***Figure 20.19.*** *Toenail cracks.*

Fluid-filled pockets may be found beneath overgrown cuticles. Sweat glands are concentrated in the skin associated with the cuticles. Overgrowth of the cuticles may put pressure on the ducts and obstruct the discharge from the glands, causing a buildup of the fluid. Elephant caretakers call these "blisters" or "blebs." Fluid pockets may rupture and become infected. Management of the fluid pocket requires returning the cuticle to normal architecture.

Routine toenail maintenance includes trimming the cuticle and thinning the interdigital skin, if necessary, by use of a hoof rasp or shaping with a hoof knife. Severely overgrown cuticles may be trimmed with an equine hoof nipper, but it may be necessary to cut the cuticle back in stages to avoid hemorrhage of the skin.

Contusion of the toenail. A blow to the surface of the toenail may produce a contusion or hematoma of the laminae, similar to the injury caused by hitting one's fingernail with a hammer. Excessive exercise with elongated toenails may also traumatize the nails.

Soaking the feet in cold water may help stop extravasation of plasma or blood and soothe pain. Administration of an analgesic, such as butorphanol, may also be indicated. See Chapter 15.

**Sole (slipper, pad).** The external keratinized layer that produces the flexible slipper on the bottom of the elephant foot is formed from a deep germinal epithelium. The arrangement is similar to that of the sole in the horse. Growth is from 0.5 to 1.0 cm per month. If the sole does not wear sufficiently, it becomes thickened, and because the thickening is seldom uniform, defects are produced that lead to pocket formation and overgrowth, which sets the stage for infection. The result may be a condition similar to thrush in and around the frog of a horse's hoof.

Other problems include foreign body penetration, lacerations, development of cracks or fissures, and generalized maceration with softening of the keratin. All these predispose to subsole abscessation or pododermatitis.

Clinical signs are similar for most conditions, including lameness, reluctance to move, and flinching when pressure is applied over an inflamed area. In more severe cases, there may be swelling, heat, erosions, ulceration, and granulation tissue proliferation. Tetanus is a potential sequel to abscessation.

Abrasion of the sole. If thinning of the sole is noticed, watch the elephant's behavior. If closely observed, it may be determined that the elephant constantly turns in a specific location and in the same direction (stereotypic behavior). This causes excessive wear on a specific area of the sole.

Another predisposing factor is a conformational fault or an injury that causes the elephant to walk in such a manner as to produce excessive wear on a segment of the slipper. An elephant may become habituated to pawing with one foot, which may wear a toenail and the sole excessively. A shuffling gait brought about by arthritis may also produce uneven wear.

Creativity is required to solve this problem, possibly including changing the floor surface, redirecting activities, and using protective devices on the foot, such as repeated application of duct tape or the use of a sandal.

Trimming the sole. The sole is a broad, relatively flat surface, which makes trimming with conventional equine or bovine hoof-trimming tools difficult; however, persons with responsibilities to provide routine maintenance of elephant feet will have a variety of tools from which to choose. It is wise to learn how to use tools that are available. Tools for elephant foot care include a hoof knife, hoof rasp, hoof groover, Buffalo brand hoof groover and knife combination, Xacto knife (X router blade, No. X161, a grooving blade), drawing knife (spoke shaver), equine hoof nipper, electric rotary grinder, Swiss cutting knife, curette, stiff-bristled brush, rat-tailed file, fine bastard file, and sharpening stone or hone (Fig. 20.21).

Power grinders are generally not recommended except for skilled, experienced foot caregivers. Grinders speed

**Table 20.4.** Antimicrobial Agents Applied to Lesions of the Foot

| Generic Name | Trade Name | Source | Indications | Mixing Directions | Comments |
|---|---|---|---|---|---|
| Copper sulfate $CuSO_4$ | Bluestone, blue vitriol; a blue granular powder | Garden supply store, pharmaceutical company | An astringent in dilute solution | As an astringent, a 1.0% solution (10 gm/l of water | Caustic in concentrated solution |
| Chlorine-dioxideoxychlor complex | Ciderm liquid and gel | ARCO Research Inc., SUNY Farmingdale, Conklin Hall, Farmingdale, NY 11735 | A powerful oxidizing agent; also an excellent antimicrobial and deodorant | Use as supplied | |
| Dilute acetic acid | Vinegar, 5% acetic acid | Grocery store | Used as an antimicrobial and cleansing solution | Use as supplied or dilute to 1% (200 ml 5% vinegar to 800 ml water) | Glacial acetic acid is 36–37% acetic acid and is caustic |
| Dimethylsulfoxide | DMSO | Veterinary supply companies | Used as a solvent to facilitate movement of other medications into tissues | Use as supplied | Causes a disagreeable odor in the breath of the animal; wear rubber gloves |
| Formalin, 10% formaldehyde | Formalin | Veterinary supply companies | Powerful disinfectant | 2.5% solution (250 ml of 10% formalin to 750 ml water) | Quite caustic |
| Hydrogen peroxide ($H_2O_2$); colorless, odorless liquid | Peroxide, hydrogen peroxide, 3% solution | Any drugstore or pharmacy | Powerful antiseptic when in contact with tissue fluids, causing foaming and cleansing | Use as supplied | Do not inject into puncture wounds or into closed cavities |
| Ammonium ichthyolsulfonate, bitumen sulfonatum, ichthammol | EquiPhar | Vedco | Slightly irritant; draws abscesses to a head, reduces swelling and is somewhat antiseptic | 20% ointment; a mixture of a product of the distillation of bitumen with lanolin and petrolatum; contains 10% sulfur | |
| Copper naphthenate, 37.5% | Kopertox | Fort Dodge Laboratories, Fort Dodge, IA | Wound protectant and disinfectant; apply daily following cleansing of the wound | Use as supplied | Can be removed from hands and clothing with lighter fluid |
| Zinc oxide (ZnO) | Zinc oxide (ZnO); a white to yellowish-white powder | Veterinary supply companies | Antiseptic, soothing, will protect skin from exudates | Made into a 20% ointment | Ointments may attract dirt and debris |
| Potassium permanganate $KMnO_4$ | Potassium permanganate | Mallinckrodt Chemicals | Antiseptic | 1:1000 concentration (1 g $KMnO_4$ in 1 l water) | Rarely used in current veterinary medicine |
| Sucrose | Granulated sugar | Any grocery store | Hydroscopic, will desiccate organisms, stimulates wound healing | Apply as supplied | Sugar has been used effectively in both human and animal wound treatment |
| Polyhexosamine polymer from deacetylation of chitin | Chitosan flakes | Vanson Inc., Redmond, Washington | Stimulates wound healing | 1% chitosan and 1% glacial acetic acid in water | |

the work and diminish the labor involved in trimming, but if they are used unwisely, the sole and toenails may be trimmed excessively and sensitive tissue be overheated.

A critical factor is the correct sharpening of cutting tools. Hoof knives and groovers need to be sharpened on the inside of the hook.

For trimming the surface of the slipper, the author recommends the use of a drawing knife (spoke shaver), which is a nonflexible blade mounted between two handles (see Fig. 20.14). Drawing knives have limited use in modern woodworking, so it may be necessary to special-order one from a hardware store. The drawing knife should be pulled steadily toward the operator. It is easy to cut too deeply and expose the corium. One should

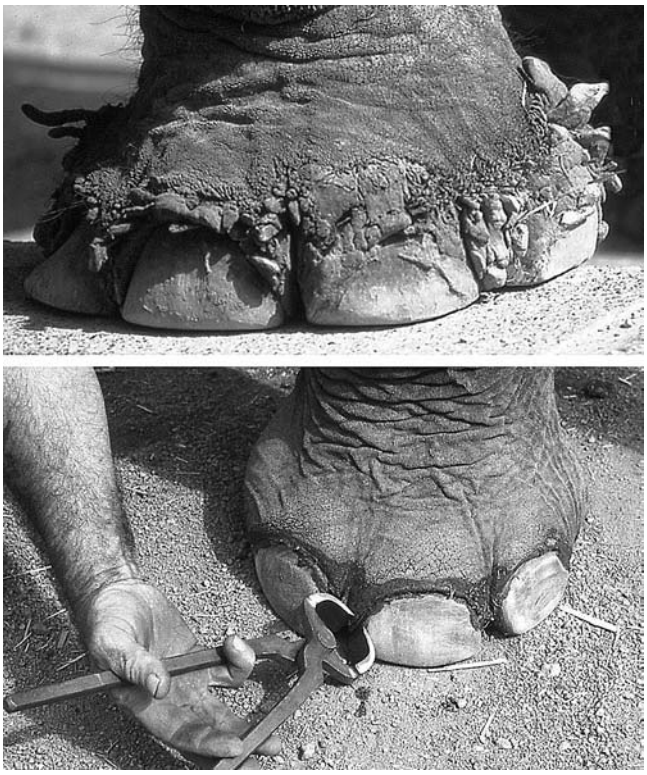

***Figure 20.20.*** *Cuticle overgrowth (hangnail). Top: numerous hangnails; Bottom: trimming with an equine hoof nipper.*

trim cautiously, shaving off one thin sheet at a time. Sight of a yellowish-pink keratin indicates closeness to the corium.

Severely overgrown soles may present a mosaic of ridges and grooves, setting the stage for numerous pockets. Normal soles are from 4–12 mm thick, varying with individuals and the amount of activity. Grooves should be beveled to discourage lodging of debris, but overzealous trimming of the sole should be avoided. It is not necessary or desirable to eliminate all the grooves and ridges. The drawing knife should be used in conjunction with a sharp hoof knife to pare out pockets and grooves to healthy keratin. Black tracts must be eliminated, and all chalky sole should be removed. Severely overgrown soles may require frequent, repeated trimming to return the foot to a healthy condition without cutting into the corium. However, the greatest mistake made in foot care in elephants is not being aggressive enough in providing drainage for infected pockets on the sole surface. An elephant that has had overgrown soles for a considerable time may walk daintily following trimming.

Contusion (bruise) of the sole. A contusion results from a rupture of blood vessels in the corium beneath the sole. Predisposing factors include stepping on a stone or other object that becomes a point source when weight is applied to the foot, walking on gravel or broken-up pavement, walking in muddy areas in cold climates that have frozen clumps of mud, and excessive trimming of the sole. The author has watched circus elephants during a walk to the performance site that walk slowly on questionable surfaces, watching where they place their feet, and pick up the pace when the surface is smooth.

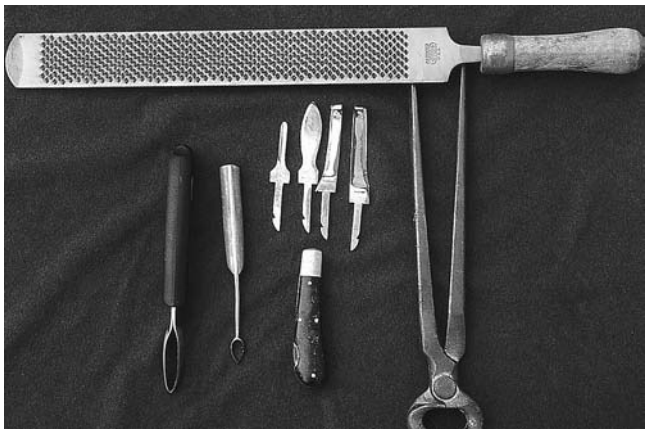

***Figure 20.21.*** *Tools used for pedicure of elephant feet.*

Clinical signs may or may not exhibit lameness. Evidence of a contusion on the slipper surface may not appear until the sole has grown out, leaving a reddish stain. If a contusion is suspected, digital pressure may cause the elephant to flinch. A contusion may result in a seroma beneath the sole and may become infected, causing an abscess.

Treatment of mild contusions may be unnecessary. If digital pressure finds an extremely sensitive spot, the elephant should be rested from any walking activity and the foot checked daily. The area over the sensitive region may be shaved carefully to relieve pressure, but the corium should not be exposed. Nonsteroidal analgesics may be used for a few days (less than a week). If an abscess is suspected, an ultrasound examination of the sole may disclose a pocket of exudate, in which case the abscess must be drained.

Subsole abscesses. Abscesses may be focal or diffuse. Severe diffuse subsole infection may undermine the sole, necessitating removal of a section of the sole or in extreme cases most of the slipper. The infection is usually located between the slipper and the germinal epithelium, so a new keratinized sole will form once the infection has abated.

Ultrasonography may be used to determine subsolar abscesses and undermining.[41] Remove as much of the detached sole as possible to provide adequate drainage and penetration of disinfectant solutions. Protect the sensitive corium with dressings—or better still, a boot.

## Musculoskeletal System

**Trauma.** The bones of the foot may be traumatized by other elephants or, more commonly, by tethering acci-

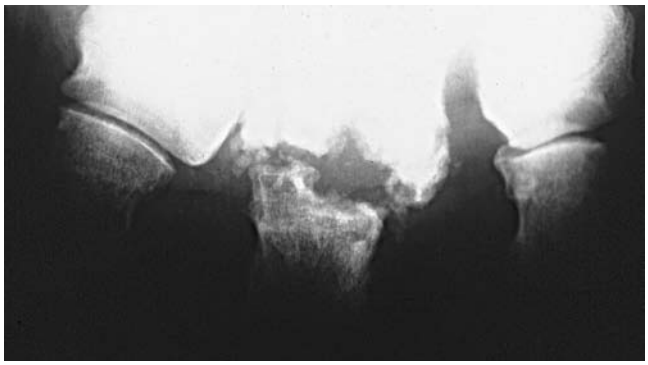

***Figure 20.22.*** *Osteitis of the phalanges.*

dents. Clinical signs may be dramatic because the elephant may refuse to put weight on the limb. Heat and swelling may or may not be evident. Radiography should be considered in a nonresponsive lameness that apparently involves the foot; see SECTION II, "Radiography," of Chapter 13.

Unfortunately, it is not possible to place the foot in a cast because of foot expansion when weight is exerted on the foot. Extended restriction of exercise is recommended. Analgesics may be indicated, but overuse of these may encourage the use of the limb when the elephant should be resting.

**Septic osteitis (osteomyelitis).** Elephant limb bones do not have a marrow cavity,[55] so technically they don't develop osteomyelitis. Septic osteitis of the phalanges is the most serious condition affecting the foot (Fig. 20.22).

Predisposing factors. Penetration of a foreign body into the bone (glass, nails, wire, wood sliver) may result in osteitis. More commonly, osteitis occurs as a result of improper management of a soft tissue infection in the foot that spreads to adjacent bones.

Clinical signs. Lameness may be subtle or pronounced. Usually there is a draining fistula near one of the toenails.

Diagnosis. Survey radiographs of the feet should be on file for comparison. When septic osteitis is suspected, the foot should be reradiographed for evidence of bone involvement. Usually P-3 and P-2 are the phalanges involved.[18,23] Particular attention should be paid to P-3 because this bone may have variable normal appearance and may appear as a thin horizontal sliver of bone with fractures present[17]. Osteitis will present with the typical starburst degeneration of bone. A fistulogram may provide definitive diagnosis.

Management. It is tempting to begin a course of broad-spectrum antibiotics, but experience with osteomyelitis in other species indicates that this is futile unless the infected bone is removed surgically. The surgery is difficult, and the aftercare required is intensive and prolonged.[9,12,17,60,61]

**Infective (septic) arthritis.** Infective arthritis may accompany septic osteitis as the infection spreads proximally. It may also be caused by foreign body penetration of a joint or may have a hematogenous origin, especially in neonates. Sepsis may rapidly cause erosion and destruction of the joint cartilage.

Predisposing factors. Joint trauma may predispose to organisms that cause another disease in the elephant. Umbilical infection (navel ill) is another source. Hypogammaglobulinemia may contribute to septic arthritis in calves.

Clinical signs. Septic arthritis is one of the most painful conditions of the skeletal system. An elephant is reluctant to place any weight on the affected limb and may remain recumbent if capable of doing so. In any species, septic arthritis, fractures, and an open joint are conditions suspected when an animal refuses to place weight on a limb. Heat, swelling, and pain associated with palpation or manipulation are cardinal signs.

Diagnosis. Severe lameness should prompt the clinician to take radiographs of the foot; however, periosteal reaction and osteitis may not be evident in early stages. The definitive diagnosis may rely on aspiration of synovial fluid from the infected joint space. Aspiration should be performed aseptically so that the fluid may be cultured. Not all infected joints yield an organism because the infection is localized in the synovial villi and periarticular tissue. The fluid may be cloudy, purulent, or hemorrhagic, with decreased viscosity and an increased WBC above 33,000 per $mm^3$.

Management. The diagnosis of septic arthritis is an emergency, and treatment should be designed to eliminate the infective organism and remove the harmful products of synovial inflammation and fibrin that can damage the articular cartilage.[62] Broad-spectrum antibiotic therapy should begin immediately, even before the results of a sensitivity test are returned. Lavage of the infected joint with physiologic saline solution or other solutions via arthroscopy is being used in equine medicine.[62] No reports are currently available for lavaging a septic joint in elephants. Should such treatment be indicated, it is recommended that an equine surgeon at a veterinary school be contacted for the latest methodology.

Regional perfusion with antibiotic solutions has been used in elephants.[39,60]

**Mycoplasmal arthritis.** Mycoplasmal arthritis is discussed in Chapter 11.

**Septic tendosynovitis.** Multiple flexor and extensor tendons and associated synovial sheath are present in the foot. Trauma (contusion) to any of the tendons may cause tendonitis. Lacerations and foreign body penetrations may also affect tendons.

Predisposing factors. Tendosynovitis is usually secondary to other primary infections within the foot. It may also be a sequel to surgical removal of a septic osteitis.

Clinical signs. Lameness, heat, swelling, and tenderness to palpation are indications of tendosynovitis of the extensor tendons. Flexor tendons lie deep within the foot and are more likely to be the tendons affected secondarily. Swelling may compromise circulation, resulting in lower limb edema. Synovial fluid may drain from a lacerated tendon.

Diagnosis. Clinical signs are primary. Ultrasonography is a diagnostic tool used in equine medicine, but no reports have been published on its use in elephants.

Management. Treatment is the same as for septic arthritis.

**Degenerative joint disease (DJD, osteoarthritis).** Osteoarthritis may be considered as a group of disorders characterized by a common end stage, which is a progressive deterioration of the articular cartilage, accompanied by changes in the bone and soft tissues of the joint.[32] DJD is a major cause of disability in captive elephants and thought by some to be the result of poor management practices with captive elephants (Fig. 20.23). However, arthritis has been identified in prehistoric mastodon skeletons. As in horses, DJD in elephants is not likely a single specific disease, but different conditions that have the same conclusion. DJD may affect any of the limb joints, but in this section emphasis is given to foot joints.

Predisposing factors. This is a controversial subject. Lack of exercise, housing on hard surfaces, and tethering are frequently brought forward as causes of DJD, but aging and wear and tear (trauma from performing repetitive actions) may have a bearing on the development of articular cartilage deterioration. The elephant's bulk and lack of angulation of the limb bones promote concussion of articular cartilage. Concurrent conformation defects or injuries that cause an elephant to alter its normal gait and change the pressure patterns within joint surfaces may have an influence. Certainly this has been shown to be a factor in human DJD.[32]

Clinical signs. Nonworking elephants may or may not exhibit lameness. Astute observation may detect subtle changes in gait, or the elephant may have been noticed to be less active recently. DJD was not detected in an elephant in a small zoo in California until a previously submissive enclosure mate became dominant and knocked the elephant down and pushed her under the railing of a fence. Although extricated from beneath the fence, she was unable to rise until lifted to her feet with a crane. Radiographs then detected multiple joint DJD.

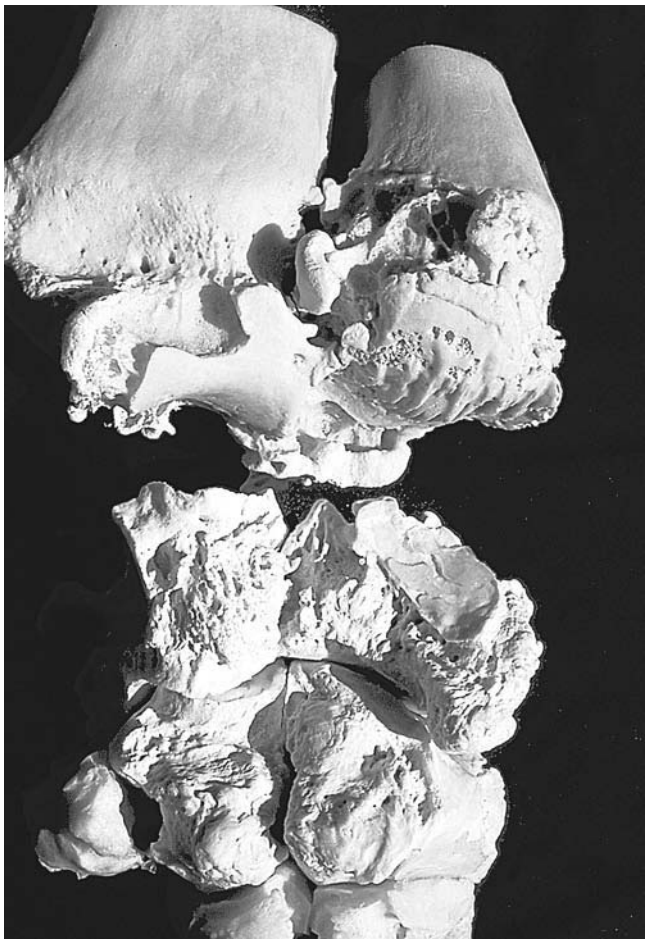

***Figure 20.23.*** *Degenerative joint disease of the elephant carpus.*

Diagnosis. Radiography is the primary means of diagnosing DJD. The characteristic radiographic findings in DJD include narrowing of the joint space, subchondral bone sclerosis, marginal osteophyte (joint mice), and periosteal bone proliferation.[32]

In acute cases, motion of a joint may be diminished (flexion of the foot joints is minimal at best, so this requires astute observation), and there may be swelling and heat over the front of the foot. Later, heat and swelling may be less evident. Arthroscopy is a definitive diagnostic tool in horses,[32] but it has not been described for elephant foot DJD.

Management. The regimens for treatment of DJD are almost as numerous as the people who implement treatment. This is understandable because the type of DJD and the stage of disease at which a diagnosis is made

vary. No sound studies for treating foot DJD in elephants have been forthcoming.

In horses, treatment is based on three principles. First, prevention or treatment of any primary cause. Second, treatment of active soft tissue disease contributing to articular cartilage degeneration. And third, treatment, if possible, of the cartilage degeneration (cartilage curettage, osteophyte removal).[32] A fourth principal might be management of pain.[4] See Chapter 15 for details. The author recommends reading the reference and/or contacting an equine surgeon at a veterinary school.

**Fractures.** Fracture of the foot bones is rare but the diminutive third phalanx may suffer multiple transverse fractures that apparently cause no clinical signs and have an unknown etiology.

**Ankylosis of joints.** Arthritic joints may become fused when the articular cartilage is destroyed and periarticular and articular bone proliferation bridges the contiguous bones. Foot bone articulations may develop ankylosis because movement is minimal. In the early stages of DJD, pain and discomfort accompany movement of the joint (Fig. 20.24). When ankylosis is complete, no pain may be associated with the joint, but stiffness may alter the gait and wearing pattern of the slipper and toenails. An altered gait may predispose the foot to other conditions.

### Parasitic Diseases

Parasitic diseases of the foot are discussed in more detail in Chapter 12.

**Myiasis.** Many species of blowflies (bottle flies, family Calliphoridae) may deposit eggs in open wounds of the foot. The larvae feed on exudates and necrotic debris and usually do little damage, but the larvae are unsightly.

Screwworm flies are another matter. The New World screw worm *Chochliomia hominivorax* is currently absent in North America. The Old World screw worm is *Chrysomya bezziana*. Both have similar life cycles. The female fly deposits eggs at the margin of fresh wounds. Second instar larvae invade living tissue, and third instar larvae bury themselves in living tissue so only the posterior peritremes are visible. Severe destruction of tissue may accompany infestation.

**Microfilarial dermatitis.** Microfilarial dermatitis around the toenail bed and heels of Asian elephants has been ascribed to a *Stephanofilaria* sp. (indistinguishable from *S. assamensis*) in Asia.[8,63] See details in Chapter 12.

## PREVENTION OF FOOT PROBLEMS

Elephant foot care involves daily inspection of the feet (stiff-bristled brush and hoof pick), exercise, training, sound nutrition, sanitation, spending as much time as possible on dirt or grass, and periodic pedicures.[49] Some elephant managers advocate providing heated floors during cold weather, which may contribute to the comfort of the elephant and hasten drying. Others disagree. Alternatives to concrete floors include heavy rubber mats, straw, or wood pallets. Exercise is more easily accomplished in a free contact program. Elephants may be taken for walks or used in elephant rides. Rides are controversial but they do address the need for exercise. It takes more creativity to encourage exercise in voluntary contact behind a barrier.

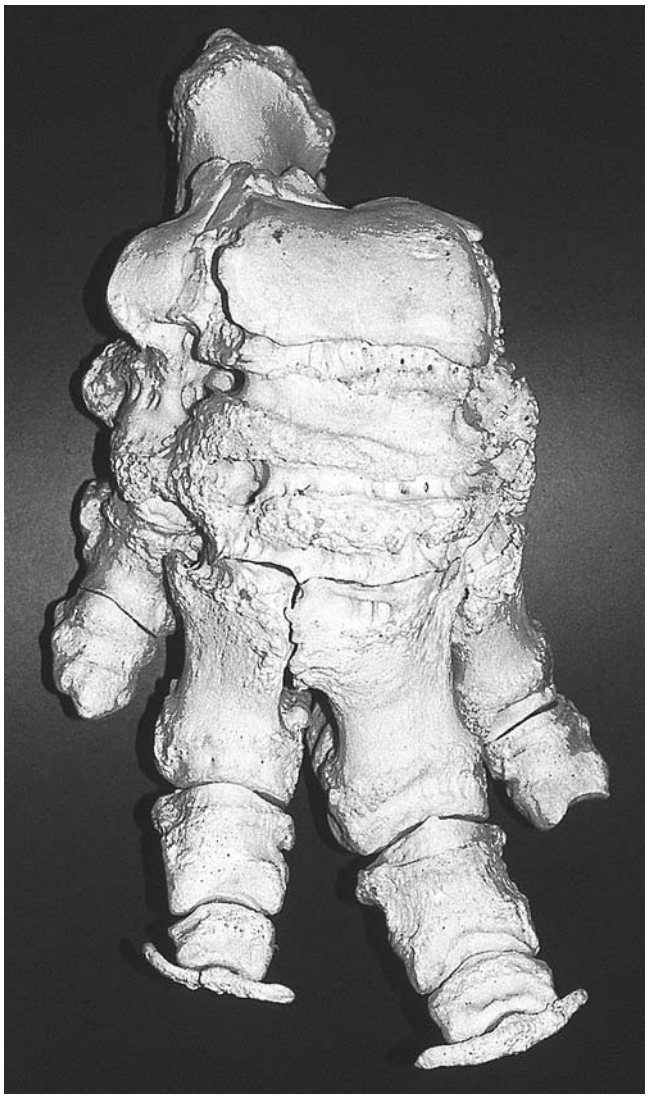

***Figure 20.24.*** *Degenerative joint disease and ankylosis of an elephant foot.*

### Nutrition

Energy intake must be correlated with body condition. Excessive weight is a detriment to foot health. Be aware of the caloric content of the diet. Free-choice hay and/or fruits and vegetables may be contraindicated for certain elephants (easy keepers). Grass hay is usually sufficient

except for juveniles and late-gestation pregnant or nursing females.[59]

Biotin (vitamin B) has been shown to be beneficial to equine hoof health.[48] No studies have been conducted on elephants, but vitamin and trace mineral supplements may be appropriate to minimize deficiencies. Essential trace minerals known to have a function in skin and nail health include zinc, selenium, and arsenic, all of which may also be toxic when used in excess. Metabolic bone disease may occur in calves.[11,36] See Chapter 6.

## CONCLUSIONS

The following conclusions can be drawn regarding foot disorders in elephants:

1. Prevention is the key to elephant foot health.
2. Training is basic to routine foot care and therapy.
3. Daily foot inspection should be routine.
4. Early detection and aggressive management are crucial to rapid healing of lesions.

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# 21 Respiratory System

Ramiro Isaza

## INTRODUCTION

As the largest surviving land mammal, the elephant possesses several unique anatomical features of its respiratory system. Despite more than 300 years of observation and scientific writings, there are relatively few publications regarding the anatomy and physiology of the elephant respiratory system.[27] Francis G. Benedict undertook a 15-month study of an adult female Asian elephant in an attempt to document the comparative physiological function of elephants.[3] His detailed observation of one elephant has been the primary source of information about the physiologic function of elephants for seven decades. In this chapter a review of the literature, a discussion of the significance for the clinician, and when necessary, the limits of our understanding of the respiratory system of elephants will be presented.

## ANATOMY AND PHYSIOLOGY

### Trunk and Nasopharynx

The trunk is probably the most unique and characteristic anatomical feature of the elephant. It is used for tactile investigation, prehension, alimentation, olfaction, and respiration. It is formed from an elongated combination of upper lip and nose.[34] Elephants are not obligate nasal breathers and can routinely voluntarily breathe from their mouths, especially when the trunk is placed in a breathing apparatus.[3]

The trunk is devoid of bone and is primarily composed of muscle, fibrous connective tissue, and skin.[34] See Figure 21.1. The paired nasal passages are roughly symmetrical and completely separated throughout the trunk and nasopharynx by a continuous, fibromuscular nasal septum.[35] They remain more or less constant in size and shape for approximately the distal three-fourths of the trunk length. They are lined by moist squamous epithelium until they enter the skull (personal communication, Dr. S. Terrell, Orlando, Florida, 2005). In the distal section of the trunk the nasal passages are very resistant to collapse from external forces and generally do not expand or contract an appreciable amount.[5]

In the proximal fourth of the trunk the anatomy and function become much more complex. See Figure 21.2. The nasal passages form a complex sinuous course and change in both cross-sectional appearance and volume. The nasal passages move from their position in the center of the trunk and run deeper into the trunk, until they parallel the cranial surface of the concave-shaped premaxillary bone. Slightly below the level of the eyes they turn rostral toward the cranial surface of the trunk, where they enlarge in volume and are compressed medially by two large muscles.

After this expansion, the nasal passage diameters decrease as they bend caudally to enter the skull.[36] This area corresponds to the bulge on the surface of the trunk that is often seen moving while the elephant is drinking.[36] At the entrance to the skull, the nasal passages may be completely closed by a combination of cartilage and muscles.[22]

The nasal and mesethmoid cartilages originate from the vomer and nasal bones to form a portion of the nasal septum and two cartilaginous wings that run over the dorsal aspect of the proximal naris.[8,35] The combination of the nasal cartilages, expanded volume of the upper nasal passages, and a complex set of facial muscles form the distensible alinasal pouches allow the elephant to hold fluid in the external trunk.[22]

It is unknown whether elephants have the ability to control the function of each nasal passage independently. However, there are some observations that elephants who have sustained a penetrating injury to one nasal passage can draw water up the contralateral passage (personal communication, Dr. J. Shoshani, Bloomfield Hills, MI 2001).

At the beginning of the nasopharynx the passages become relatively small and narrowed by several turbinates.[19] Here the lining of the nares changes from squamous epithelium to smooth mucosa, more typical of the upper respiratory tract of other species (personal

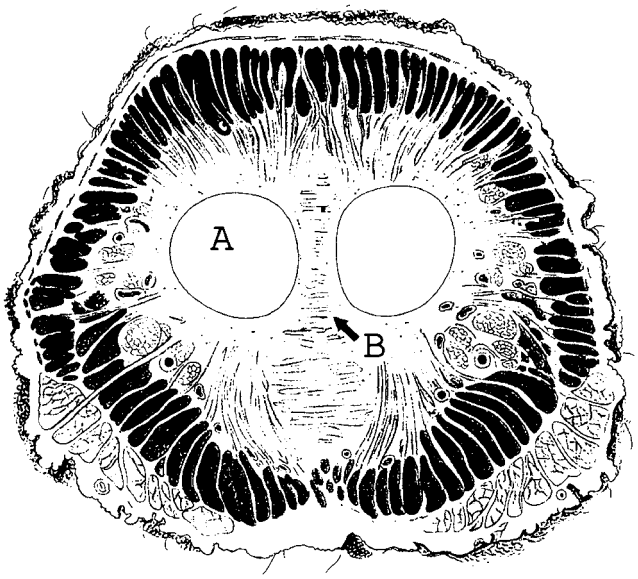


***Figure 21.1.*** *Drawing of the cross section of the distal trunk. Paired nasal passages (A) are in the center of the trunk and separated by continuous nasal septum (B).*

communication, Dr. S. Terrell, Orlando, Florida, 2005). Along the dorsal aspect of the cranial nasopharnyx there are multiple passages leading into the sinus systems in the skull. Elephants lack a nasolacrimal duct and therefore are presumed to lack a corresponding ductal opening into the cranial nasopharynx. Further caudally, the eustachian tubes enter the caudal nasopharynx.[19] The large maxillary teeth (molars) are located just lateral to either side of the caudal nasopharynx.

Adult elephants contain a very large, air-filled sinus system that provides the majority of the volume of the massive elephant skull.[36,37] The spongelike sinus system contains thousands of small spaces separated by thin, bony walls with a mucosal lining. The sinus system is rudimentary in young elephants and expands during growth.[36,37] The full function of this sinus system and the amount of air circulation within it has not been well documented.

## Pharynx, Larynx, and Trachea

The pharynx is a funnel-shaped tube that provides a passageway for the respiratory and gastrointestinal tracts. The rostral portion is divided into the oropharynx and nasopharynx by the short soft palate.[33] The caudal oral cavity is very narrow and partitioned by a sphincterlike opening.[33] The caudal portion of the oropharynx also has a unique pharyngeal pouch located between the base of the tongue and the front of the epiglottis.[34] This pouch is estimated to hold several liters of fluid that may be kept for at least several hours without interfering with feeding or breathing. The pharynx has glandular and lymphoid tissues in the middorsal area.[13] There are also pocketlike palatine tonsils in the lateral pharynx.[13]

The larynx consists of the usual five cartilages (thyroid, cricoid, epiglottis, and paired arytenoids) that are held together with ligaments and muscles to form a short tube lined by mucus membrane.[13,19] The epiglottis is short and thick with a rounded free border.[13,19,35]

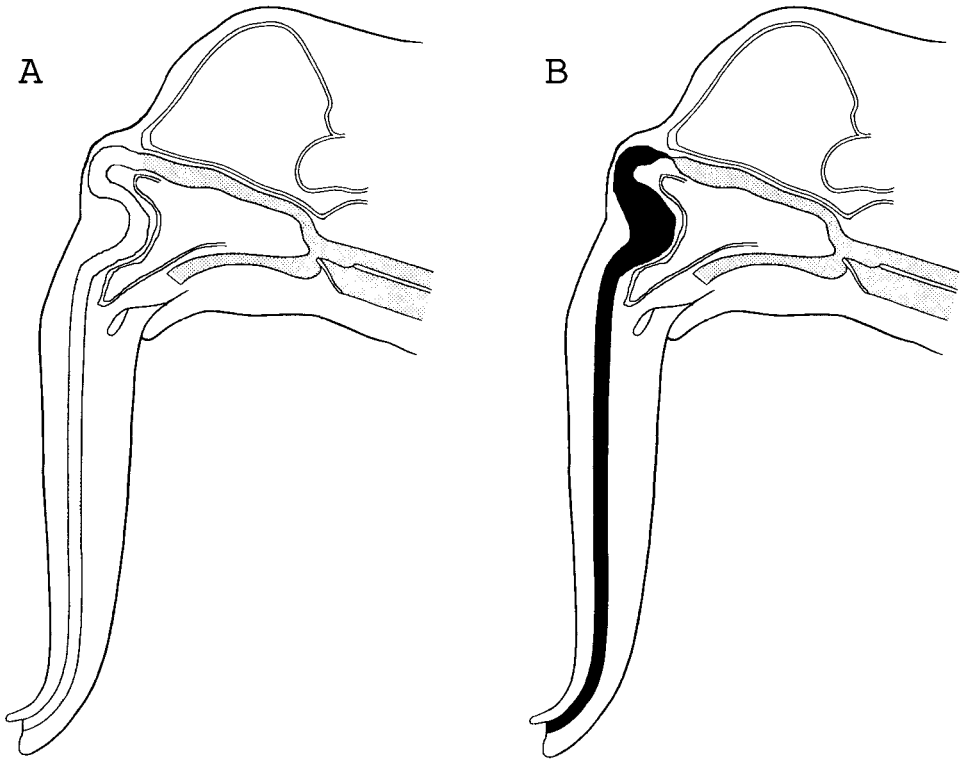


***Figure 21.2.*** *Sagittal view of nasal passages through the trunk, nasopharynx, and larynx. A) The air-filled trunk during inspiration of air, B) a similar view illustrating water in the trunk and showing the ability to close off the nasal passages near the entrance to the skull.*

The trachea begins at the cricoid cartilage of the larynx and extends intrathoracically to the bifurcation of the bronchi at about the level of the fifth thoracic vertebrae.[19] The tracheal lumen of an adult African elephant is about 30 cm long and 5 to 7 cm in diameter.[5,35] It is supported by large, incomplete, cartilaginous tracheal rings.[5,35] The dorsal end of each ring articulates with the adjacent rings, forming a "sawtooth" pattern along the dorsal tracheal surface.[5]

## Thorax

The thorax and intercostal muscles are typical of other mammals.[13] The Asian elephant has 19–20 ribs and the African has 20–21 ribs.[8,13,19,35] The thorax is shaped like a truncated cone that is compressed laterally to be deep and narrow.[19,36] Presumably due to low rib cage compliance, elephants breathe primarily by contraction of the diaphragm and displacement of the abdominal contents, rather than by expansion of the thorax.[36] This may lead to dyspnea while in a sternal position because the gastrointestinal tract may push forward and limit diaphragmatic movement. This is probably more important during chemical immobilization because the animal is unable to make necessary postural changes to ensure continued effective ventilation.

Elephants lying in lateral recumbency while sleeping or resting seem to have little problem ventilating, and they may sleep for 4–5 hours every night.[3,30]

A study comparing respiratory function in standing versus lateral elephants found no clinically relevant reduction in arterial blood oxygen content.[15] This was true despite the substantial reduction in pressure of oxygen in the arterial blood in the recumbent position, possibly due to recumbency induced ventilation-to-perfusion mismatch and/or decreased minute ventilation. Although hemoglobin saturation was decreased below that found in the upright elephant, there was a hemoconcentration secondary to the recumbency-induced arterial hypertension that effectively counterbalanced the effect of the reduced oxygen pressure and restored the arterial oxygen content to upright levels.[15]

The diaphragm anatomy is typical in configuration and relative size to other large mammals. The attachment of the diaphragm extends from the dorsal sixteenth rib to the ventral second or third rib, giving it an oblique orientation to the spinal axis.[5,13,19] The diaphragm muscle fibers generally radiate from the central tendon toward the ribs.[13] Using information from the dissection of one African elephant, it was estimated that an elephant could generate a negative pressure of approximately 200 cm water, consistent with the negative pressures needed to snorkel while swimming.[5,39]

## Lung

Generally the elephant lung is similar to most other mammalian species.[9,10] The fresh lungs of four adult Asian elephants weighed an average of 21 kg.[11] The left lung is slightly smaller than the right and consists of one lobe with several deep fissures partially separating the ventral portion of the lung. The right lung consists of one major lobe with partial fissures similar to the left and a smaller mediastinal lobe near the pulmonary hilus.[19]

The acini and alveoli inside the elastic partitions are slightly larger that other mammals and are exceptionally numerous, thus providing an extensive respiratory surface.[9,10] Except for the initial part of intrapulmonary primary bronchus, the elephant lung lacks cartilage.[5,38] Instead, the lung is subdivided into small, grapelike (1 $cm^3$) parenchymal units by a network of elastic intralung septa that originate from a much-thickened visceral pleural capsule.[5,10,38]

The visceral pleura are continuous with the intralung septal network, and similar dense connective tissue surrounds the larger airways and vascular structures in both the lungs and pleura.[10,39] This network of elastic tissue throughout the lung appears to provide support to the lung in place of the more typical mammalian cartilaginous support system.[5]

The visceral (lung capsule) and parietal pleural surfaces lack the typical mesothelial serous membranes seen in other mammals.[5] These two pleural capsules are connected to each other by a pleural space connective tissue (PSCT).[5] This homogeneous tissue is diffusely and continuously attached to all the surfaces of the inner thoracic cavity (ribs) and the diaphragm.[5] See Figure 21.3 (Color Section). The unrelated Malayan tapir (*Tapirus in-*

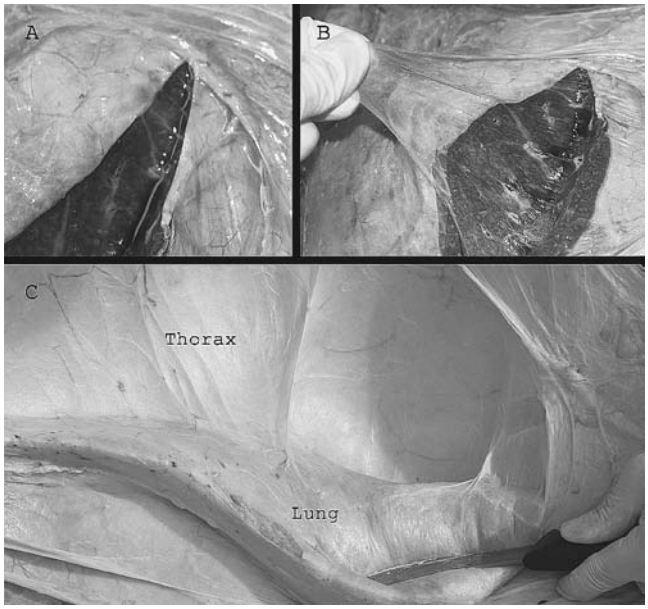


***Figure 21.3.*** *Three photographs of normal pleural space connective tissue (PSCT) from an Asian elephant. A) Close-up view of the relaxed, gelatinous PSCT resting on the lung surface; B) a portion of the PSCT is pulled away from the lung surface; C) lung is pulled and cut away from the thoracic wall, showing the extremely stretched PSCT as it is typically seen during necropsy. Also see this image with the color plates in Chapter 18.*

*dicus*) is the only other large terrestrial mammal species reported to have similar connections between lungs and chest wall.[2,17,36] Interestingly, it has been noted that the fetus of elephants, until late gestation, develops with nonadherent lungs.[39] The early descriptions of the elephant's pleural cavity suggested tight adhesions that might be expected in pathological conditions such as chronic pleuritis. These observations have led to the common belief that these adhesions were rigid and that the normal pleural function is obliterated.[36]

Currently the PSCT is described more as a loose connection between the lung and costal surfaces.[5,31] The PSCT is a 3D fibrous network filled with fluid pockets and some capillaries.[5] The tissue is noninflammatory, uniform in density, and described as deformable but not elastic.[5,38] Histologically it is composed almost entirely of collagen with only occasional elastin fibers.[5] When stretched during necropsy by the weight of the lungs, the PSCT stretches over 20–50 cm and becomes the consistency of spongy fiberglass that may be penetrated with a blunt instrument, but is more easily cut with a sharp knife.

When unstressed, as in its natural state, the PSCT has a slick, almost gelatinous consistency that is difficult to cut due its ability to deform and shear laterally.[5] The mobility and slippery consistency of the PSCT tissue suggest that it allows movement and sliding of the visceral pleura during breathing.[5] Despite its unique anatomy, the elephant may have the functional equivalent of a typical pleural space, thus negating the idea that the lungs are rigidly adhered to the thoracic wall.[5]

The combined presence of the PSCT, the thick pleural capsules, and intralung septal network have all been well documented; however, the function of these tissues has not yet been fully determined. Early speculation was that the elephant's peculiar anatomy is needed as protection from pneumothorax or pleuritis, and that their "mode of life" does not require much blood oxygenation or efficient lungs.[37]

Later, it was thought the system combined to dampen the strength of diaphragmatic contraction and thus limit the rate at which the lungs could fill (Todd 1913). Without the tight adhesions, the diaphragm would contract too fast, resulting in "pronounced diminution" of the intrapleural pressure.[38] This would potentially result in hemorrhage, damage to the lung parenchyma, or a spontaneous pneumothorax.

A more current hypothesis is that the elastic network of tissue inside the lung and surrounding the visceral capsule combine to prevent the alveoli in dependent areas of the lung from collapsing due to the weight of the lung.[5,10] Concurrently, it may prevent the upper lung from overexpanding due to stretching.[5] Additionally, because the loose PSCT is not needed to hold the lungs up or keep them inflated, it may instead act to regulate the flow of pleural fluid and keep it from pooling in the ventral aspect of the thoracic cavity.[5]

Two alternative hypotheses suggest that the generation of massive negative pressures in the lungs and thorax of elephants is the reason for unique PSCT and thickened pleura. In the first situation, elephants routinely draw water approximately 2–3 meters up their trunks while drinking, generating negative pressures of at least 200 cm water.[33] Short speculated that the PSCT was not needed to generate the pressures; however, the adhesions were necessary to prevent collapse or injury to the lungs during these periods.[33]

In the second situation, elephants also generate large negative pressures in the thorax while "snorkeling" underwater.[39] See Figure 21.4. Elephants are known to be excellent swimmers and have been documented to spend hours in deep water swimming with just their trunks above the water line.[34,39] Assuming that the ventral aspect of the lungs is over 2 m beneath the water surface, the elephants would again be subjected to pressures of greater than 200 cm water.[39] Elephants breathing while underwater must generate enormous pressures to expand their lungs against the water pressure.[39] This in itself would not demand a different lung structure but would require extraordinary respiratory muscle strength and effort. More importantly, however, the water pressure would raise the systemic blood pressure to about 200 cm water, and the luminal pressure within unsupported pleural capillaries would be expected to either rupture or leak fluid excessively into the pleural space.[39]

Both of these situations produce pressures that greatly exceed the 30–40 cm water typically generated by other mammals. In fact, during forced laboratory inspiratory maneuvers, the human respiratory system cannot generate pressures much greater than 160 cm water and can be injured when even brief attempts are made to inhale against these pressures.[39] In contrast, elephants can apparently withstand these pressures without injuring the delicate vessels in the parietal pleura. West theorized that the anatomical configuration of connective tissue surrounding the vessels in the thickened pleura functions to provide additional support to protect these vessels from damage and from fluid leakage inundating the pleural space.[39] This theory does not address the unprotected capillaries noted in the PSCT.[5] Brown suggested that some capillary leakage in the PSCT normally provides a source of pleural fluid; however, massive injury to the PSCT while generating negative pressures was not addressed by either author.[5,39]

Whether the elephant's unique pleural structures are designed primarily to protect the elephant while imbibing water or snorkeling cannot be resolved at present. It is true that snorkeling activity might be sustained longer than the brief episodes of drinking and therefore the opportunity for lung damage might be commensurately greater. However, it is also true that many elephants might not, through necessity, engage in swimming behaviors, whereas drinking is not optional.

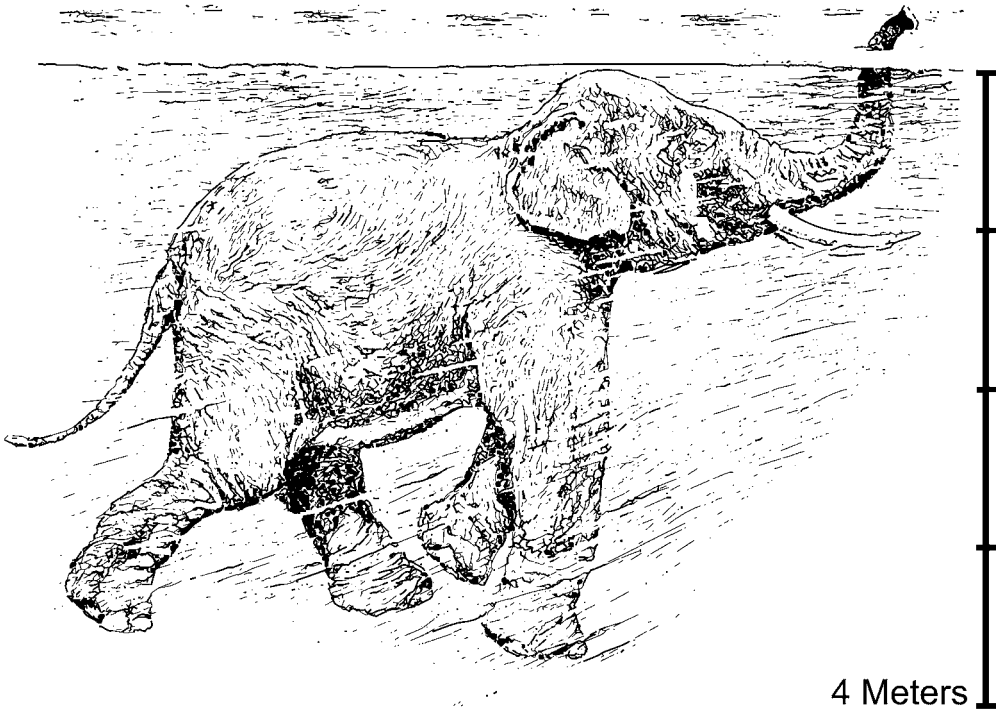


***Figure 21.4.*** *Illustration of a male Asian elephant swimming underwater and using the trunk as a snorkel. The ventral aspect of the lungs is about 2 meters beneath the water surface, subjecting the elephant to transthoracic pressures of at least 200 cm water.*

### Blood and Respiratory Function

Elephant red blood cells are the largest of all mammals with a volume that exceeds 131 $\mu m^3$.[7] Both elephant species have hemoglobin with an affinity for oxygen that is more pronounced than most mammal species.[1,7] This affinity of the hemoglobin for oxygen benefits loading of oxygen in the lung but impairs offloading in the muscle. In keeping with their great mass, elephants have a low mass specific metabolic rate of 1.1–1.8 ml $O_2$ per kg per minute,[3] which reduces the demand for oxygen offloading at the tissues compared to that present in smaller mammals, such as the human. Thus, the oxygen-binding characteristics of elephant blood are well suited for their metabolic demands.

## CLINICAL EXAMINATION

### Trunk and Nasopharynx

A limited physical examination of the respiratory tract may be performed on a cooperative animal. A well-trained elephant in a free contact management system provides the most direct access, but an elephant in a restraint device may be adequately examined. Regardless of access or apparent cooperation, working near the head and trunk of the elephant is dangerous, and appropriate precautions should be taken. Additionally, due to the possibility of tuberculosis, care should be taken when directly exposed to exhaled air from the animal.

Normal respiratory noises are not usually heard from the trunk while standing near the elephant. However sudden deep exhalations and various vocalizations can occasionally be detected audibly. The clinician may place a cupped hand over the nostril openings to feel the air flow during both phases of respiration and compare differences between nostrils. If air passage in one nostril is questionable, some elephants will allow the clinician to occlude one nostril and assess air flow in the other. The amount and character of nasal discharge should also be noted. Normal elephants have moist nostrils with a small amount of clear fluid present in the trunk tip (Fig. 21.5). Occasionally abnormal odors can be detected from the exhaled air.

Auscultation with a stethoscope on the sides of the trunk may detect air passing through the nasal passages. However, this is possible only if the elephant is cooperative and keeps the trunk still. Further proximally, at about the level of the eyes, the nasal passages pass closer to the surface of the skin and may be more clearly auscultated. These respiratory sounds in the trunk are primarily generated by air passing through the naris, and are often mixed with a variety of vocalizations.

Endoscopy and visualization of the distal nasal passages are relatively easy in a cooperative or preferably sedated elephant. In a nonsedated Asian elephant, the several bends in the upper naris and the voluntary muscular control combine to occlude the nasal lumen, making the passage of the endoscope past the proximal trunk very difficult (personal communication, Dr. D. L. Schmitt, Springfield, Missouri, 2005). In contrast, Gage reported endoscopic examinations of three sedated African elephants with no apparent difficulties noted in passing the scope into the nasopharynx.[12] It is unknown whether there are significant species variations in nasal anatomy or whether the increased level of seda-

***Figure 21.5.*** *Examination of the trunk tip of an Asian elephant. The animal must be trained and cooperative to allow manual restraint.*

tion prior to the procedure may have made passage of the scope easier in the African elephants.

### Pharynx, Larynx, and Trachea

The structures of the pharynx, larynx, and trachea are difficult to examine in the conscious elephant. Visual examination of the oropharynx is prevented by the large molar teeth and a narrow caudal oral cavity. The ventral aspect of the larynx and proximal trachea may be superficially palpated through the skin of the ventral neck. Having the elephant extend the head and neck provides better access, but the examination is still limited by the massive, short, muscular neck.

### Thorax

Measurement of respiratory rate during a physical examination in a normal standing elephant is challenging due to the very limited thoracic excursions and constant movement of the animal.[3] However in a quiet and cooperative elephant, the frequency of breathing may be measured by careful visual inspection of thoracic and abdominal excursions over 90-second intervals. The normal standing respiratory rate for elephants is 4–6 per minute.[30] While in lateral recumbency, the breathing pattern has greater thoracic excursions and the respiratory rate can be discerned with ease.[15] The normal respiratory rate for sleeping elephants is 3–8 per minute.[3]

### Lung

Clinical signs of pulmonary disease are often subtle, but may consist of fever, increased respiratory effort, discharge from the trunk, and occasional coughing.[11,24] Due to an elephant's size, clinical assessment of the lungs in elephants may be difficult. Thoracic wall thickness and the musculature of the front leg prevent effective pulmonary auscultation. Radiographs of the chest are possible only in very young elephants using powerful, stationary radiographic units. Passage of an endoscope into the lungs is probably possible only in the fully anesthetized, recumbent elephant. The trunk wash technique is the recommended method of collecting samples for mycobacteria culture in standing elephants.[16]

### Blood and Respiratory Function

The color of the tongue and mucosa of the vulva can be used to assess the oxygenation of the blood. An anesthetized adult Asian elephant was used to demonstrate the clinical usage of pulse oximetry attached to the ear and end-tidal $CO_2$ from the trunk.[23] The large vessels located behind the ears may be used to collect arterial and venous blood samples for blood gas determination.[15] Blood gas analysis has been reported from both standing and recumbent nonsedated elephants.[14,15,23]

## DISEASES

### Trunk and Nasopharynx

A catarrh or coldlike syndrome has been reported to occur in Asian elephants.[11,30,37] Signs included constant discharge of clear, watery fluid from the trunk tip, swollen eyes, loss of appetite, and listlessness. The cause was undetermined, but the signs are similar to upper respiratory viral infections in other species. Supportive care and rest was generally curative; however, a secondary bacterial pneumonia may develop during convalescence.

Septic inflammation of the sinuses (sinusitis) caused by penetrating trauma or "sunstroke" has been described.[37] Clinical signs of fever and furious behavior presumably caused by pain were also noted.[37] Effective treatment of an infected sinus system with either antibiotic therapy or surgical drainage would be difficult due to its complex structure, limited potential for drainage, and sheer volume of the sinus system.

The nasopharynx is the narrowest portion of the upper respiratory tract and is therefore subject to pathologic stricture. A progressive left-sided nasal occlusion caused by a benign osteoma in the paranasal area of the skull has been seen in an adult Asian elephant (personal communication, Drs. G. Kollias, Ithaca, N.Y. and C. Wallace-Switalski, Pittsburgh, PA, 2005). Similarly, a unilateral partial obstruction of the right nostril was noted in another Asian elephant caused by an impacted upper molar that pushed medially into the nasopharynx (personal communication, Dr. S. Terrell, Orlando, FL, 2005).

Traumatic injury to the distal trunk is rarely documented in the literature, but lacerations and crush injuries have occurred. One published report described

the amputation of the distal eight inches of the trunk of a young African elephant. Surgical reattachment was considered impractical; however, surgical reconstruction of the remaining trunk tip was attempted.[6] Primary surgical closure and healing would be difficult due to the mobility of the trunk and the ability of the elephant to rub the surgical site.

### Pharynx, Larynx, and Trachea

Swelling in the parotid area and pharyngitis have been reported in elephants in Asia.[11,37] Large swellings between the mandibles or under the neck can cause respiratory difficulty due to partial obstruction of the pharynx and larynx. Although presumed to be caused by bacterial infections, these descriptions did not include bacterial isolation or identification. Two additional fatal cases of pseudomembranous pharyngitis were mentioned in a German textbook.[29]

Nematode gapeworms, *Mammomonogamus loxodontus* and *M. indicus*, have been found in the trachea and bronchi.[35,36] Larvae of the elephant bot fly, *Pharyngobolus africanus,* have been noted throughout the upper respiratory tract of wild African elephants.[35,39]

A food allergy resulting from hay was described in an African elephant.[12] Clinical signs consisted of dyspnea and abnormal respiratory sounds. An endoscopic examination revealed severe swelling of the pharyngeal tissues and larynx. The elephant responded to diet modification, antibiotics, and dexamethasone treatment.

An adult Asian elephant developed clinical signs of pharyngitis and rhinitis following inhalation of chlorine gas. During the course of routine barn cleaning, a cloud of gas was generated from a fire produced from the inadvertent mixing of swimming pool powdered bleach and an organic material, probably mineral oil, that had been left in a bucket. Within 24 hours the animal was markedly depressed and had a trunk droop, profuse salivation, and anorexia. The elephant also developed bilateral corneal edema. The condition resolved slowly over a 2-week period (personal communication, Dr. D. Heard, Gainesville, Florida, 2005).

### Thorax

Blunt or penetrating injuries to the thorax that resulted in fractured ribs have been briefly described in elephants from Burma.[11] Tusk injuries from fighting could potentially injure the chest but are rarely documented.

**Lung.** Tuberculosis in elephants has been known to exist for 2000 years and is currently the most important respiratory disease of captive elephants.[25] *Mycobacteria tuberculosis* is the most common pathogenic organism isolated from infected elephants.[25,29] The clinical signs of tuberculosis in elephants may include weight loss, anorexia, weakness, and dyspnea. However, elephants are usually asymptomatic. See Chapter 11 for more detailed information.

Primary pneumonia and bronchitis caused by a variety of bacteria that are commonly associated with pneumonia in other species have been described in elephants.[11,24] As an example a young African elephant died with *Pseudomonas pneumonia* and renal failure (personal communication, Dr. S. Terrell, Orlando, Florida, 2005). Often the antemortem diagnosis of bacterial pneumonia is based on clinical signs, and treatment consists of parenteral antibiotics and supportive care.[30]

Isolation of environmental atypical mycobacteria was reported from 68.6% of 442 trunk wash samples and generally considered to be nonpathogenic.[25] However, in debilitated animals, mycobacteria such as *M. elephantis* and *M. szulgai* may be isolated from elephants with significant lung pathology[32] (personal communication, Dr. S. Terrell, Orlando, Florida, 2005). Herpesvirus has been noted in the lungs of a high percentage of otherwise healthy wild African elephants.[20] Fungal infections are incidentally mentioned in the literature but are apparently rare.[24]

One case of aspiration in an African elephant described pulmonary congestion and secondary bacterial infection after inhalation of plant material.[21] Two young African elephants foraged in a garden and apparently died from cardiac glycoside poisoning with lung congestion after eating palay rubber vine (*Cryptostegia grandiflora*).[4] See Chapter 33 for more details. Fibrosarcoma that began in the foot of a 54-year-old captive Asian elephant had multiple metastases to the lungs.[18]

Several important elephant diseases such as anthrax, endotheliotropic herpesvirus, encephalomyocarditis virus, and hemorrhagic septicemia (*Pasteurella*) may produce pulmonary lesions but are not generally considered primary pulmonary pathogens.

### Blood and Respiratory Function

The elephant's myoglobin has a sixfold greater affinity for carbon monoxide than human myoglobin.[28] This suggests a potentially lethal susceptibility to carbon monoxide toxicity, and consideration should be given when housing elephants in enclosed areas heated with combustible fuels or during transport where exposure to automobile exhaust is possible.[26] To date there have been no case reports of carbon monoxide toxicity.

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# 22 Digestive System

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## ANATOMY

### Teeth

The elephant dental formula is I 1/0, C 0/0, PM 3/3, M 3/3. The tusks are extensions of the upper second incisors. Young elephants develop deciduous tusks (called *tushes* in African elephants) that grow up to 5 cm in length. Tushes consist of a crown, root, and pulpal cavity. Although the tush has no apparent function, it provides the foundation and orientation for the development of the permanent tusk.[35] Tusks are a prominent feature of both male and female African elephants, *Loxodonta africana,* and male Asian elephants, *Elephas maximus.* Female Asian elephants may also have small vestigial tusks (also called *tushes*).[9,10]

The tushes are replaced by permanent tusks within 6 to 12 months of age. The tusks emerge from a sulcus on either side of the caudal upper lip, near the commissure. Permanent tusks grow approximately 17 cm per year and continue to grow throughout the life of the elephant.[40] Male tusks can grow 6 times faster than female tusks. Elephants are presumed to have a dominant (working) tusk, either on the right or the left, as evidenced by a difference in the degree of wear between the two tusks.[26]

Tusks are used to dig for water or strip bark from trees when browsing. They are also used to break branches when browsing and to carry heavy items. Males may use tusks as weapons during conflicts.

Most of the tusk is composed of a combination of hard, elastic calcium salts and dentin. Inside the tusk is a noncellular matrix. The central tract is composed of secondary dentin extending from the apex throughout the tusk length. The pulp is composed of blood vessels, nerves, and lymphatics.[40] See Figure 22.1.

Through the use of histochemical staining with S-100 protein antibodies, nerve bundles have been identified in most portions of a transected tusk pulp in a young African elephant. Nerve bundles were identified both closely associated with and independent of vessels.[4] The pulp is loosely attached to the canal and is easily damaged or removed.

An elephant will have 24 molar teeth in the course of its lifespan, each arcade containing 6 teeth. Premolars and molars consist of enamel and dentin arranged in a number of lamellae. Each lamella is bonded to the next by cementum. Cementum covers the external surface of each molar with the exception of the apical part of the crown.[40] See Figure 22.2. Many authors and scientists use the term *molar* to describe both premolars and molars in the elephant. For the remainder of this chapter, the term *molar* will be used to designate teeth that are not incisors (tusks).

A newborn has two or three small teeth in each arcade. Molar teeth erupt in the caudal jaw and move forward progressively over time. Molars may be identified, with experience, by their size and gross appearance. The approximate age of an animal at the time of shedding of particular molars has been studied fairly closely. In African elephants, molar I is lost between 1–2 years of age, molar II at 3–4 years of age, molar III at 9–10 years, molar IV at 19–25 years, and molar V at approximately 45 years. Molar VI is the last molar in the arcade and will remain whole or in part for the remainder of the elephant's life. See Table 22.1.

When molars come into wear, the pattern of loops and convolutions form. These are classified as *lamellae* or *laminae*. The enamel loops of the African and Asian elephant differ in shape. In the African the loops are lozenge shaped on the occlusal surface; the Asian elephant has simplified closed loops. The Asian elephant has a greater number of loops per molar than the African. The average number for each molar, upper and lower, for both species (African/Asian) is 5/5, 7/8, 10/13, 10/14, 12/19, and 13/24 for molars I to VI, respectively.[40] See Chapter 1 for illustrations.

Molar teeth are worn down and shed in sections, and they are replaced by the next tooth pushing forward from behind. This drifting of the teeth provides contin-

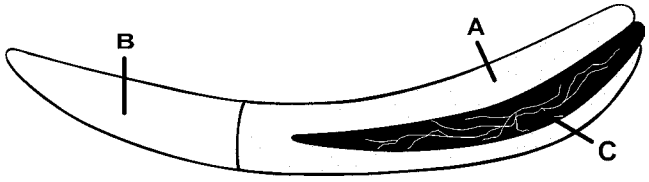


***Figure 22.1.*** *Side view cutaway of an erupted tusk. The wall of the tusk encircling the pulp cavity (A). Distal end of the tusk (B). Vessels and nerves are roughly diagrammed within the pulp canal (C). Modified from McCullar, 1994. Illustration by R. Green.*

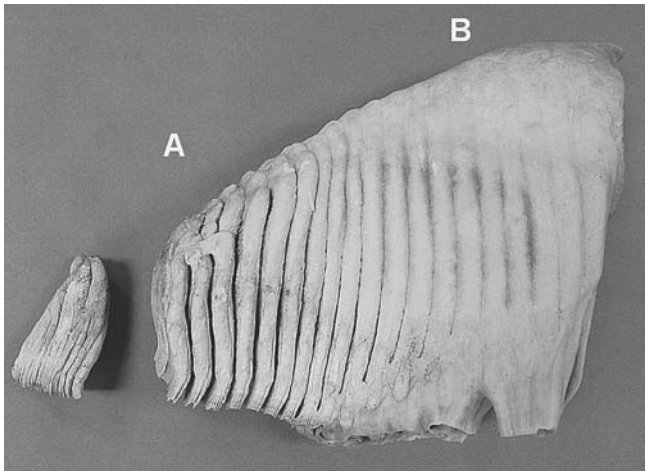


***Figure 22.2.*** *Side view of a developing and erupting upper molar of an adult Asian elephant taken postmortem. At left the individual lamellae are visible stacked against each other (A). At right the lamellae are becoming covered with cementum that binds them together into a solid molar tooth (B). The roots of this tooth were broken off when the tooth was removed postmortem.*

**Table 22.1.** The Molar Number with Its Corresponding Number of Lamellae, the Approximate Eruption Time for Each Molar, and the Approximate Age that Replacement of Each Tooth Occurs.*

| Molar # | Number of Lamella | Eruption | Replacement |
|---|---|---|---|
| 1 | 4 | 4 months | 2–2.5 years |
| 2 | 8 | 6 months | 6 years |
| 3 | 12 | 3 years | 9 years |
| 4 | 12 | 6 years | 25 years |
| 5 | 16 | 20 years | 50–60 years |
| 6 | 24 | 40 years | 60+ years |

Modified from Kalita 2003.

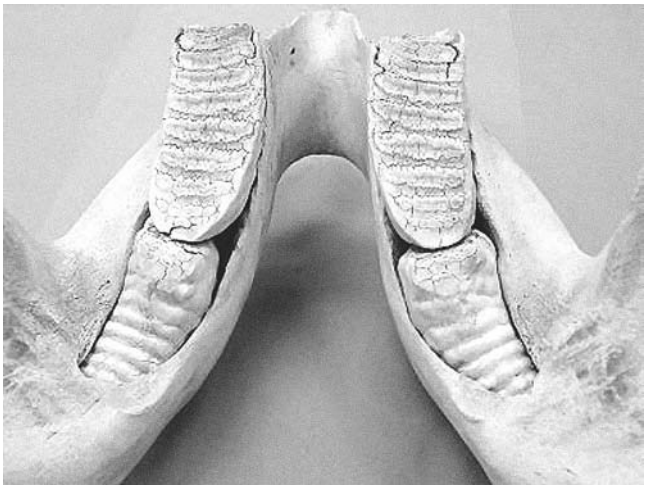

***Figure 22.3.*** *Lower molars of an adult Asian elephant, showing a normal plane of progression and delamination. The laminae are visible on the occlusal surfaces of the rostral teeth. Note the rostral aspect of the caudal molars coming into wear.*

uous contact. Each new tooth is larger than the preceding one. Each adult molar tooth may weigh over 5 kilograms. Discarded tooth fragments may be found as they fall out of the mouth or may be found in feces after being swallowed. In many instances the fragments are not found.[23,40]

Several methods of aging elephants using dental characteristics have been described and critiqued.[21,23,24,25,37,42] Some methods of aging both Asian and African elephants employ the appearance and migration patterns of the molars. The number and appearance of lamellae have also been used to age individuals. Sike's method proposes the use of the relationship between the anterior lamellae of the tooth in wear on the right mandible relative to its location to the foramen mentale.[42] In contrast, Laws proposes ages based on a method involving a shoulder height/age equation.[25] Jachmann has compared and contrasted both methods and proposes that the methods of Sikes may be more valid, and when employing the method of Laws, different sets of data should be available for each habitat type.[19] Objective assessment of age based on tooth morphometrics may be obtained from lamellar counts and length and width measurements. Teeth I, II, and VI seem to be the most distinctive morphologically when fully developed. Molars V and VI seem to be the most difficult to differentiate between based on appearance and size.[19] However, in the intact jaw, molar VI will have no molar developing behind it. See Figure 22.3.

As the molars progress forward they reach the foramen mentale in the mandible. It is at this point and beyond that tooth root resorption begins. As the roots continue to resorb and the tooth progresses forward, the anterior portion fragments from the more caudal, viable portion of the tooth.[42]

The trunk tip of the elephant is designed for prehension of all types of food. The tip is dexterous enough to pick up small seeds and can even pick up an apple by its stem. Asian elephants have a single projection on the top of the trunk often referred to as the "finger." The

lower portion of the tip is flat. In the African elephant the trunk tip possesses opposable projections on the top and the bottom. Both species show similar dexterity despite the anatomical differences. The trunk is then rolled ventrally and the food is placed directly into the mouth.

## Oral Cavity

The oral cavity is small relative to the size of the elephant. The upper lip combines with the root of the trunk. The lower lip narrows to a point in front. The mouth is very narrow with a large, fleshy, dexterous tongue. The tongue is unable to protrude from the mouth due to the attachment of the tip of the tongue to the floor of the mouth. The tongue can form a fold in the center that aids in directing food into the back of the mouth. The trunk is used to pick up and deposit food and water into the back of the mouth.

There is essentially no difference in the superficial architecture of the Asian and African elephant tongues. Many anecdotal reports and experience by the author indicate that elephants have preferences for different foods and flavors. Taste is discussed in Chapter 32.

Caudal to the mandibular ramus and condylar process bilaterally lay the parotid salivary glands. They are below the external auditory meatus and deep to the zygomatico-auricularis muscle. The glands are related deeply to the digastricus muscle, facial nerve, and posterior auricular artery in the Indian elephant.[28] In the adult African elephant they have an average weight of 7.4 kg. The glands are of the tubulo-acinar type. There are slightly elongated homocrine acini containing seromucous type secretory cells in each gland.[34] African elephant saliva does not contain alpha-amylase. Therefore a digestive role of saliva is likely negligible; however, saliva may serve as a lubricant for coarse ingesta. Levels of calcium, magnesium, phosphorous, potassium, and urea are higher in saliva than serum. Elephant saliva contains little protein, no albumin, and no sodium. Salivary composition may vary with age, hydration, hormonal status, and salivary secretory rate. The elevated urea content of saliva may be indicative of a recycling mechanism in which protozoa and bacteria use urea for metabolic processes.[34]

On the side of the caudal oral cavity at the posterior one-third of the nasopharynx is a pharyngeal diverticulum. It is located just caudal to the pharyngeal opening and is capable of holding less than a gallon of fluid. The boundaries of this pouch are as follows: The floor extends from the epiglottis forward. It is formed from behind forward by the thyroid cartilage, thyro-hyoid membrane, and the hyoid bone. The lateral walls are completed by the sides of the pharyngeal muscles, the stylo-glossi and hyo-glossi muscles. The root of the tongue forms the anterior boundary, and the posterior wall is completed by a depression of the soft palate. When the soft palate is elevated, the pouch communicates freely with the esophagus.[31] The pharyngeal diverticulum may function as a fluid reservoir between the stomach and the oral cavity and may explain the anecdotal observation that elephants can withdraw fluid from their stomachs.

## Stomach

The stomach of the elephant is simple, with numerous rugal folds in the cardiac portion. The cardiac sphincter is very thick and muscular. The caudal third of the stomach has a smooth mucosa.[28] The volume of the stomach of an adult female Asian elephant has been reported as a maximum of 76.6 l.[39] The average volume of the stomach of 10 adult African elephants was 60 ± 5 l.[45] An adult bull stomach has an empty weight of approximately 36–45 kg.[43] That of an adult Asian female is 17.35 kg.[39]

## Liver

The elephant liver has 2–3 lobes. Sikes reports three lobes to be more common. The right lobe is largest when two lobes are present. Individual variation is thought to be responsible for the difference in the number of liver lobes. The liver weight in an adult cow averages 36–45 kg and in adult bulls averages 59–68 kg.[43] There is no gall bladder but biliary canals are present. The hepatic duct joins with the major pancreatic duct and then enters the wall of the duodenum forming an ampulla containing numerous mucosal folds.[28]

## Pancreas

The pancreas is transversely elongated in the mesoduodenum. It is both exocrine and endocrine and in the adult weighs approximately 2 kg.[28] See Chapter 23.

## Intestines

The small intestine is comprised of the duodenum, ileum, and jejunum. Several reports indicate that the length of the elephant small intestine ranges between 11 m to 21.6 m.[32] The differences in length are likely due to variations in size and age of the individuals. Shoshani[39] determined the volume of the small intestine of an adult to be 133.56 l. There are a few glands and Peyer's patches present in the duodenum. On the lateral wall of the duodenum is the hepatic ampulla, which forms from the hepatic duct after receiving the major pancreatic duct. The minor pancreatic duct opens at this same level on the medial wall.[28,31]

The duodenum begins at the pylorus of the stomach and ends at the jejunum. The jejunum is held in a coil by the mesentery in the floor of the abdominal cavity located on the left and right sides.[28] The last section of the small intestine is the ileum, which ends at the cecum. It opens into the cecum at the ileocecal orifice.[28]

The large intestine includes the cecum, colon, and mesocolon, which is the posterior 2 meters of the digestive tract. Shoshani[39] determined the volume of the large intestine to be 483.2 l. Several reports state the

length as between 6 m to 12.8 m.[32] The combined length of the small and large intestine may reach a length of up to 35 m.[40] A study of 10 adult African elephants states a total length from stomach to distal mesocolon as 27 m.[45]

The colon is held by the mesentery in folds in the left and right abdomen, overlying the small intestine. Two rows of sacculations are along the colonic walls. Two muscular bands lie on the walls of the colon. In the dorsal part of the pelvis the colon becomes the rectum. The two muscular bands from the colon continue onto the anterior rectum. Two rows of sacculations occur on the walls of the cranial rectum. The caudal rectum has no sacculations. The rectum opens to the exterior as the anus located below the root of the tail. Pudendal nerves and arteries supply the muscles of the anus.[28] See Figure 22.4.

## DIGESTIVE PHYSIOLOGY

Elephants are hindgut fermenters. The cecum is the first important fermentation site in the digestive tract. The average cecal volume of 10 adult elephants was found to be 90 ± 10 l.[45] The colon is regarded as the main digestive area of the elephant. The colonic volume of the average adult is 329 ± 30 l.[45] The digestive strategy of the elephant is that of passing large amounts of low quality forage through its GI tract in a short period of time. Elephants in the wild spend an average of 16–20 hours a day eating.

Adult African elephants need 150–200 liters of fresh water daily.[13] In contrast, Asians have been reported to drink 200–255 l per day taking in 50–60 l at a time 3–4 times daily.[10] In the horse the maintenance fluid requirement varies from 50–100 ml/kg/day. If this amount is applied to a 4000 kg elephant, the daily fluid requirement should be between 200 l and 400 l. The actual maintenance amount of fluids for an individual varies with activity, environmental condition, and physiologic status.

Elephants are generalized feeders, feeding on brush and tree branches, bark, palms, seedlings, and a variety of grasses. In captivity elephants are typically fed cultivated hays, concentrates, produce, and browse. For more information, see Chapter 6. Hackenberger and Atkinson[17] determined that the feed intake as a percentage of body weight was 1.03 for the Asian elephant and 1.60 for the African elephant. Gastrointestinal (GI) transit times depend on the type of foods eaten. A diet of concentrates and hay has a more rapid rate of passage than a strictly hay diet. There are also differences between African and Asian elephants. African elephants seem to pass food more rapidly than Asians. African elephants fed timothy hay and hay cubes are reported to have a mean retention time (MRT) of 22.8 ± 2.1 hours. Asians fed timothy have an average MRT of 26.6 ± 0.4 hours.[18,36] A study of elephants fed grass hay found MRTs of 50–54 hours. Animals fed alfalfa hay showed

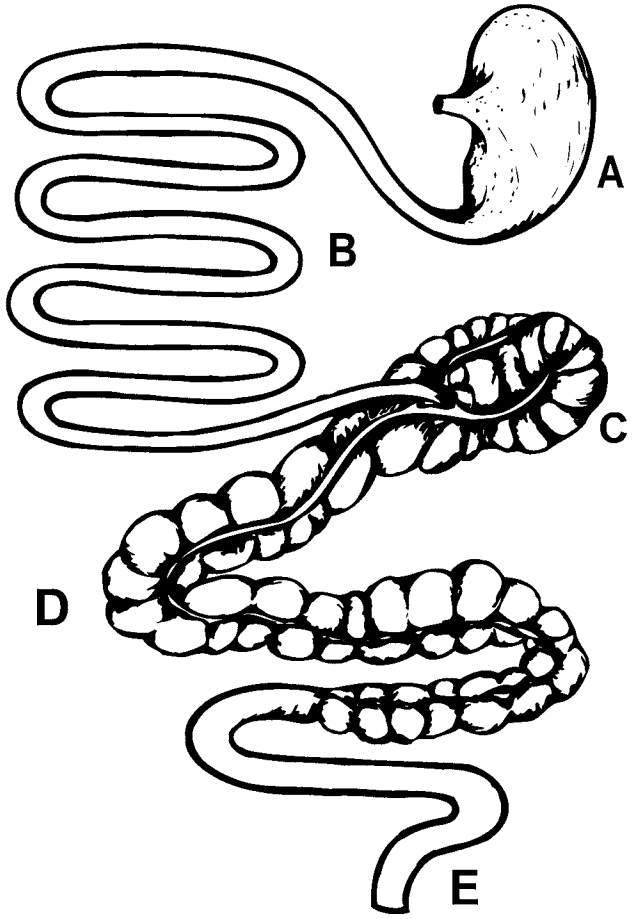


***Figure 22.4.*** *This diagram depicts the arrangement of the elephant gastrointestinal tract from the stomach to the anus: stomach (A), small intestine (B) (comprised of duodenum, jejunum and ileum), cecum (C), colon (D) (as both sacculated and nonsacculated portions), and the rectum (E). Modified from Clemens, E.T. and Maloiy, G.M.O. 1983. Nutrient digestibility and gastrointestinal electrolyte flux in the elephant and rhinoceros. Comp Biochem Physiol 75A 4:653–658.*

MRTs of 38–48 hours for Africans and 51 hours for Asians.[15] African elephants fed grass hay and straw had passage times of up to 46 hours.[36]

A more recent study investigated the passage rate of chromium oxide through the digestive tract of Asian elephants. The average rate of passage of the chromium oxide was 24 hours at first appearance and 54 hours to last marker excretion. Recovery of the chromium was 97%.[27]

Elephants may defecate as many as 15–20 times daily. The average number of fecal balls (also referred to as *boli*) is 5–8, with each ball weighing approximately 1–2.5 kg.[10] In a study of African elephants at the Tsavo National Park, the average weight per defecation in an adult was 10.36 kg. The defecation rate among four animals ranged from every 1.09 hours to 3.33 hours.

Rees[36] reported on the phenomenon of synchronous defecation in African elephants. This is a type of allelomimetic behavior. Evidence was found that elimination may be influenced by herd behavior and mutual stimulation. This synchronous behavior likely has adap-

tive significance in the wild. Elephants assume a vulnerable position when they stop to defecate, with hindlegs spread apart and tail raised. In this stance elephants cannot walk as they defecate. A young elephant in the back of the herd that stops to defecate independent of the adults would soon fall behind the herd and become vulnerable to predation. In captivity elephants have been trained to defecate on command. This provides evidence that defecation may be under regular voluntary control.

The chemical composition of elephant feces was studied in a 9-year-old male African elephant in Kenya. Components were reported as a percentage of dry matter. Mean composition of the feces was crude protein 6.92 ± 0.21, nitrogen 1.108 ± 0.033, crude fiber 46.93 ± 1.16, calcium 2.04 ± 0.085, phosphorus 0.246 ± 0.013, sodium 0.143 ± 0.008, and potassium 0.577 ± 0.025.[12] A high crude fiber fraction of almost 50% correlates well with the typical dietary items of free-ranging elephants. Details of the nutritional needs and concerns of elephants can be found in Chapter 6.

## DISORDERS OF THE DIGESTIVE SYSTEM

### Tusks

The tusks are particularly prone to injury due to their protrusion from the maxilla. The pulp is loosely adhered to the wall and is easily damaged. A crack or a hole in the tusk that exposes the pulp canal may lead to infection. A small black spot on the end of a tusk may be an indication that an infection is present. Some elephants with dental disorders will display signs of pain. Other animals with significantly damaged tusks have shown no obvious indication of discomfort. Problem tusks may have foul odors or purulent discharge indicative of chronic infection. A devitalized pulp canal or uncontrolled infection is an indication for extraction. If not treated properly, pulpitis may result in chronic infections and may be fatal.[30] Pulp infections may persist for years. Infections may involve aerobic or anaerobic bacteria, including *Streptococcus* sp., *Staphylococcus* sp., *Pseudomonas* sp., *Proteus* sp., *Klebsiella* sp., *Enterococcus* sp., *Corynebacterium* sp., *Fusobacterium* sp., *Bacteroides* sp., and *Actinetobacter* sp.[1,29.30]

Open pulp canals should be protected from environmental contamination to prevent infection or to facilitate treatment of existing infection. Elephants can readily remove caps or other devices and have a tendency to pack open canals with debris and mud.

Serious infections require aggressive therapy with antibiotics, repeated flushing, and a device to protect the canal between treatments. The use of a set screw as a removable seal worked in one animal until the infection was resolved. Then the screw was permanently placed at the opening of the canal to prevent contamination into the treated area. Over time tusk growth pushed the tract distally to help heal the damage.[51]

Pulpotomies, bands, caps, systemic and local antibiotics, and tusk flushing have all been used with success in the treatment of a variety of tusk injuries.[46,47] The degree of success depends on the severity of the injury or infection and the method used. Treatment duration may last several months.[1,6,46,49,51] Conservative management may be successful when initiated early and diligently. In one case resolution of the damaged tusk took several years.[46]

In chronically infected or severely damaged tusks, extraction may be indicated. Several techniques have been described for this procedure.[1,5] Methods include gouges and chisels to separate the tusk from the alveolar wall. Another method described sectioning the tusks longitudinally to facilitate removal.[46] In a case involving a subadult female African elephant, removal of a nonviable, infected tusk was accomplished using a series of rubber elastic bands positioned apically around the tusk at the attachment between the tusk and the sulcus wall. These bands were then regularly pushed toward the root until the tusk was exfoliated. Eventually the tooth loosened and fell out. There was significant root resorption of this tusk prior to exfoliation, and the sulcus healed without incident.[44]

The alveolar socket must be protected from contamination and trauma during the healing process. Daily flushing of the socket and packing with saline or dilute povidone iodine–soaked gauze is recommended. In some cases the socket may be successfully sealed with zinc-oxide-eugenol or calcium hydroxide. Healing by granulation occurs over several months.[46,49]

### Molars

Periodontitis may occur at the tusk or the molars and is often a result of a foreign body—such as straw, twigs, or hay—becoming entrapped between the tooth and the sulcus. Flushing and systemic antibiotics or topical antibiotic ointments are usually recommended.[49] Problems with malformed or retained molar teeth may cause periodontal issues and may prevent proper forward progression of other molars. This may also result in failure of the molars to fracture off properly, resulting in difficulty in chewing, weight loss, poor condition, and colic. Providing the animal with large hard objects to mouth and chew on may facilitate the fracturing and removal of retained molar fragments.

Malocclusions may impede or alter the normal forward progression of newly emerging molars. Impaired forward progression may delay or inhibit proper delamination of the molars. This may result in malformed or fused molar teeth that may cause chronic problems. Inspection of the oral cavity on a regular basis may help detect such problems early. See Figure 22.5. Teeth that do not shed properly on their own and cause problems for the animal may need manual extraction.

Some teeth may be so badly malformed that root resorption and thus fragmentation and loss of the teeth

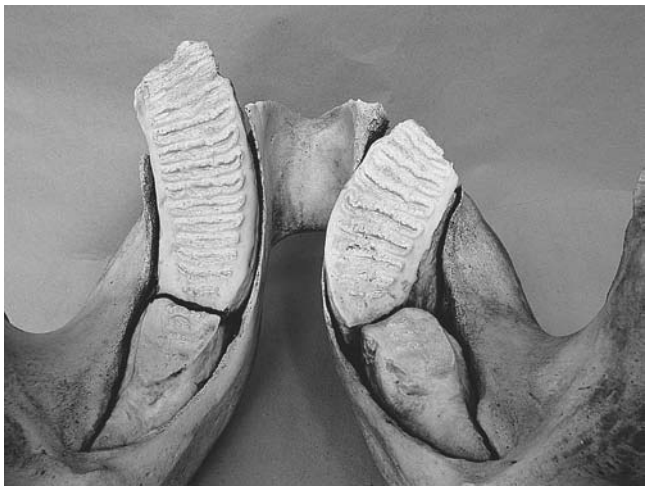

***Figure 22.5.*** *Malocclusion molars of the jaw of an adult Asian elephant. The plane of migration is abnormally curved, and the front of the molars have not fractured off properly.*

***Figure 22.6.*** *Severely malformed and fused upper molar(s) contributing to severe malocclusion in an adult female Asian elephant. The roots at the bottom of this picture were fractured off when the tooth mass was removed from the maxilla postmortem. (Courtesy R. Green)*

do not occur properly. Postmortem examination of one elephant revealed 2–3 teeth fused together in the maxilla with a molar on each side of the maxilla located almost perpendicular to the plane of progression. Malocclusions this severe may result in difficult mastication, transient-to-severe digestive problems, and oral pain. See Figure 22.6.

The oral cavity is susceptible to a variety of injuries and infections. Stomatitis has been reported in Asian elephants. This condition may result from impaired digestion, irritation to tissues of the oral cavity, oral trauma, or contact with potentially caustic substances. In the very young hand-raised elephant, thrush (candidiasis of the mucous membranes of the mouth) has been reported as a cause of stomatitis.[38] Clinical signs of stomatitis include inappetence, painful mastication, ptyalism, foul mouth odor, possibly dehydration, and constipation.[2,14] Treatment consists of removing the source of irritation or trauma and rinsing the mouth regularly with clean water or a dilute chlorhexidine solution if the elephant will tolerate this. Offer soft foods only, avoiding rough items such as hay and browse until the condition resolves. Adequate hydration should be ensured.

## Liver

Despite the lack of a gall bladder, there is a biliary system in the elephant liver. Cholelithiasis has been reported. The bile ducts were thickened and dilated grossly, but inflammation was not observed in this case. *Salmonella london* was isolated from culture broths.[11]

Parasites have been identified in the biliary tracts of elephants.[1,7] Details on parasites can be found in Chapter 12.

Liver damage may result from toxins and medications. Hepatic insult occurred in an elephant given pyrazinamide at the recommended prophylactic dose following exposure to *Mycobacteria tuberculosis.* Pancreatic enzymes were also elevated, probably secondary to liver damage. This animal recovered with discontinuation of medication and supportive care, including a reduced concentrate diet. Details on infectious diseases such as tuberculosis can be found in Chapter 11.

## Esophagus

Esophageal obstructions have occurred in Asian and African elephants.[13,41,50] Clinical signs include dysphagia (difficult swallowing), regurgitation, and weight loss. Dehydration and dry feces may also be expected if the impaction is severe enough to prevent swallowing water. Possible complications of esophageal obstruction include aspiration pneumonia, temporary or prolonged esophageal dysfunction, stricture, diverticulum formation, necrosis, and perforation. Contrast radiography is not feasible in the adult elephant. Esophageal endoscopy is a more effective means of diagnosis, although sedation or general anesthesia would usually be required. Treatment by means of esophagotomy, as well as complications from such a procedure, have been described.[41] Sikarski suggests treating the esophagotomy incision as an open wound and recommends cutting produce such as apples and oranges into sections as obstructions may result from eating whole fruit. When an esophageal obstruction is successfully removed, esophagitis should be assumed as a complicating sequela and appropriate treatment begun. Administration of broad-spectrum antibiotics covering aerobic and anaerobic organisms and an antifungal agent are recommended. Inflammation and pain at the site of trauma may be treated with flunixin meglumide or phenylbutazone. The author uses these agents no longer than 3 consecutive days to avoid problems with renal insult and/or gastric ulceration. Adequate hydration must be ensured

during therapy. The use of corticosteroids to minimize stricture formation is controversial.[16]

### Stomach

Antemortem recognition of gastritis and gastric ulceration has not been reported in the elephant. Due to the anatomic and physiologic similarities between elephants and equids, extrapolation of these conditions from the horse may be useful. Clinical signs of ulceration include poor appetite, poor condition, colic, and changes in attitude.[33] Other conditions typically precede the development of gastric ulcers. In the horse, the common diagnostic modalities are contrast radiography and endoscopy. Diagnosis in elephants may be primarily based on history, clinical signs, and clinical pathology findings that would correlate with pain, and acute or chronic blood loss.

Treatment should include H2 antagonists such as cimetidine or ranitidine. Omeprazole can effectively block gastric acid secretion but is encapsulated and must be given intact for maximum effectiveness. Sucralfate may be used in conjunction with H2 antagonists in an attempt to cover and protect the ulcerated areas.[33] Elephant dosages for the above medications are not available, but administering these medications at the recommended equine dosage may be effective.

### Intestines

Serious intestinal infections may be rapidly fatal. *Salmonella* sp. infections in the intestines of elephants result in rapid dehydration secondary to watery, bloody, mucoid diarrhea. In some cases there is scant to no fecal production but abdominal pain is evident. Ingestion of soil occurs in some cases. The small and large intestines may undergo extensive necrotic ulceration of the mucosa, which is evident as fibrinous material in the feces. Salmonella infections are considered by many to be the leading cause of diarrheal disease in elephants.[20] Treatment with broad- spectrum antibiotics and fluid therapy to correct electrolyte imbalances is indicated as early in the clinical disease process as possible.

*Clostridium perfringens* infection has been reported in an African elephant in Switzerland. The elephant was treated with antibiotics, fluids, glucose, and corticosteroids. The animal was later euthanized after becoming recumbent and unable to rise. *Clostridium perfringens* beta 2-toxin was responsible for ulcerative enteritis leading rapidly to recumbency in this animal.[3] Further details on infectious agents of the intestines can be found in Chapter 11.

Intestinal obstructions or partial obstructions are frequently a result of ingestion of dirt, clay, or sand that becomes impacted in an area of the GI tract.[48] Too much high fiber food in a short period of time may also cause obstructive problems. Obstructions of this type may lead to colic. Many of the clinical signs of colic resemble those seen in horses. Signs include restlessness, stretching, rolling or assuming a bowing posture, abdominal distension, lack of defecation, biting the trunk tip, groaning, and tenesmus. If left untreated, dehydration ensues rapidly due to reluctance to drink. Dehydration is quickly followed by intestinal stasis, electrolyte imbalance, endotoxic shock, and circulatory collapse. The condition may quickly result in recumbency and death. The earlier an obstructed animal is recognized, the more likely treatment will be successful. Treatment involves administering pain-relieving medications such as flunixin meglumide or butorphanol tartrate. If the animal is willing to eat upon diminishment of pain, a mixture of mineral oil and bran may be helpful in relieving the obstruction and stimulating evacuation. Warm water enemas may be helpful in relieving distal obstructions.

Intestinal torsion has been recognized in a few elephants in the past 15 years in the United States. Diagnosis is postmortem. Death is rapid secondary to endotoxic shock and cardiovascular failure. The causes of the torsions were not identified. In one case the animal was found to have severely maloccluded and fused molar teeth bilaterally.

Intussusception of the small intestine has been described in one case involving an Indian elephant. Diagnosis was made on postmortem examination. This animal was also found to have a heavy parasite infestation. It was surmised that the parasite infestation caused increased peristalsis, which could have led to the intussusception.[8]

Surgical intervention may be indicated in some cases of severe colic. The risks of abdominal surgery in an ill elephant should be carefully evaluated. More details on surgical conditions in the elephant are covered in Chapter 10.

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# 23 Endocrine and Immune Systems

Linda J. Lowenstine

## INTRODUCTION

The information in this chapter has been garnered from the literature and from personal observations of necropsies performed at the Veterinary Medical Teaching Hospital, School of Veterinary Medicine, University of California Davis (UCDVMTH). For each of these systems, anatomy and function will be reviewed, along with reported diseases and information about clinical assessment for each of the components of these systems.

## THE ENDOCRINE SYSTEM

Most of the literature on the endocrine system of elephants is concerned with reproductive hormones for the diagnosis of pregnancy and timing of manipulated reproduction. Endocrine aspects of reproduction are covered in Chapter 28.

### Anatomy and Physiology

As in all mammalian species the endocrine organs consist of pituitary (hypophysis), pineal gland, thyroids, parathyroids, adrenals, gonads, the endocrine pancreatic islets of Langerhans, and the diffuse neuroendocrine system (sometimes referred to as the APUD system (amine precursor uptake and decarboxylase).

**Pituitary.** The elephant pituitary occurs at the base of the hypothalamus. It consists of a pars tuberalis, pars distalis, and pars nervosa. Both Asian and African elephants, like humans, are said to lack an intermediate lobe (pars intermedia).[32] In a study of a large number of culled African elephants, Li and coauthors[23] reported an average pituitary weight of 2.5 grams; Johnson[16] reported 3.1 gm.

Several pituitary hormones (growth hormone, prolactin, ACTH, TSH, LH, FSH) have been described and compared to those in humans and domestic animals.[23,24,25,26,30] Because both acidophilic and basophilic cells occur, they have been ascribed the same function as in other species. To date, localization of the hormones by immunohistochemistry or laser capture to specific cell types has not been detailed in elephants.

Serum levels of adrenocorticotrophic hormone (ACTH) and thyroid stimulating hormone (TSH) have been measured in culled African elephants.[13,14] Reported values for TSH for African elephants are 2.10 ± 0.43 to 3.62 ± 0.53 μU/ml, and for ACTH 13.3 ± 3.2 to 39.3 ± 6.4 pg/ml. The higher values for ACTH were from elephants that were herded prior to being culled by overdosing with succinyldicholine.

Few elephant pituitary lesions have been described, but Johnson reported small cystic or follicular structures containing mineralized concretions in many animals.[16] Similar cysts were seen in a stillborn African calf and a 57-year-old female Asian elephant that also had an acidophil adenoma of the pars distalis (UCDVMTH). Acidophils in other species produce somatotrophin and prolactin, but there was no clinical or pathologic evidence that the adenoma was secretory. Mikota[32] reported on two animals that died with suspected pituitary dysfunction secondary to inflammation (old abscess and granulomas) in the region of the pituitary. Clinical details were not available for these two animals. Pituitary abscess syndrome has been described in many species of domestic animals. The abscesses arise from direct extension of septic meningitis secondary to infections from oral cavity, dehorning, and nose ring placement or through septicemic spread to the vascular rete.[35] Similar pathogenesis would be expected in elephants.

**Pineal.** Elephants, like most vertebrates, have a pineal gland.[15] Based on studies in numerous species, the main role of the pineal is in regulation of circadian rhythm and production of melatonin. It is unknown whether the pineal gland of elephants secretes melatonin, or how circadian rhythms are controlled in elephants.

**Thyroids.** Grossly, the thyroid glands in elephants have been variously described as being separate paired struc-

tures with or without an isthmus or a single fused bilobed structure. In adult elephants thyroid glands are often lobulated by varying amounts of dense connective tissue. Thyroid weight in one male African elephant was reported to be 680 gm.[36] In an older female Asian elephant, the thyroids were 11.3 × 4.5 × 3.5 cm and 62.0 g and 10 × 5.2 × 2 cm and 48.5 g (combined weight 110.5), and in another aged female Asian the left was 12.7 × 6.0 × 3.9 (not weighed) and the right was 16.5 × 6.3 × 4.3 cm and weighed 49 gm. In a 39-year-old Asian female, the thyroid gland was described to be "bilobed and reticulate" and weighed 112 g (UCDVMTH).

Thyroid glands of elephants are composed of follicles, which secrete thyroid hormone, and interfollicular C-cells, which secrete calcitonin. Calcitonin is responsible for calcium homeostasis in many, but not all, species of vertebrates. It lowers blood calcium through increasing urinary excretion and decreasing bone resorption, thus antagonizing the effects of parathyroid hormone. It is not known whether calcitonin is important in calcium homeostasis in elephants.

Clinical assessment of thyroid function relies on measuring thyroid hormone levels and response to stimulatory hormones such as TSH, along with evaluation of clinical signs and serum chemistry profiles. Abnormalities in thyroid hormone levels may occur with physiologic changes and non–thyroid-related illness, so care must be taken in interpretation. Brown[6] evaluated total serum thyroxin (T4) levels and thyroid binding globulin (TBG) T3 uptake using commercially available kits designed for use in humans. They reported good cross-reactivity and compared differences in T4 and T3 uptake in animals culled during the wet or dry season and in two different locations. Thyroxin/T4 ranged from 119.0 ± 22.8 to 174.9 ± 28.3 and T3 uptake from 135.9 ± 5.5 and 143.9 ± 7.2 nmol/l. There were significant differences in T4 between groups and wider variation between individuals within groups. In a more recent study, RIA was used to evaluate free T3, free T4, total T3, total T4, and TSH in cycling and noncycling female Asian and African elephants.[8] There were no significant differences between the two species nor were effects of stage of reproductive cycle noted. The mean values from all elephants in the study were TSH O.69 ± 0.15 ng/ml, free T4 0.94 ± 0.02 ng/dl, total T4 10.73 ± 0.24 µg/dl, free T3 1.56 ± 0.12 pg/ml, and total T3 123.97 ± 2.62 ng/dl. (Note differences in units between the two studies.)

No reports were found of either hypo- or hyperthyroidism in elephants. One paper suggested that elephants, because of their size, have a high requirement for iodine.[33] Iodine deficiency in most species leads to the formation of hyperplastic goiter and, later, colloidal goiter. Lesions reported in thyroids of elephants in the UCD files included interstitial fibrosis (which may be normal), marked variation in the size of follicles and amount of colloid, cysts, and follicular epithelial lipofuscinosis. Diffuse colloid distension of follicles was noted in a stillborn African elephant. The significance of this change was uncertain, but it was not noted in the thyroids of other calves from the same contributing institution.

**Parathyroids.** In our experience at UCD, elephant parathyroids may sometimes be difficult to find during a routine necropsy. In African elephants they are described to be paired structures, two on each side, associated with the ventral margin of the thyroid.[46] In Indian elephants a superior and middle pair are described and reported to measure 3 × 5 and 2 × 2.5 cm in a calf.[28] Parathyroid hormone (PTH) is responsible for maintaining serum calcium through increased intestinal absorption of calcium, decreased urinary excretion of calcium, and increased release of calcium from bone through osteoclastic resorption. The effects of PTH are antagonized by thyrocalcitonin secreted by C-cells of the thyroid. Parathyroid hyperplasia would be expected to occur in the face of chronic renal disease, nutritional imbalance of Ca:P, or the lack of vitamin D.

Reported clinical conditions associated with abnormal calcium metabolism include hypocalcemic tetany and rickets. Hypocalcemic tetany has been reported in elephants housed without exposure to natural sunlight and as a result of stress of transport.[40] Tetany has also been reported in elephants housed indoors in the absence of full spectrum sunlight.[41] Rickets is a metabolic bone disease in young animals due to Ca:P imbalance, and it has been suspected in elephants.[40] Parathyroid lesions noted at necropsy included a parathyroid adenoma in a 24-year-old female African elephant with dystocia, and marked fatty infiltration was noted in an older Asian cow. The adenoma was apparently nonsecretory, because hypercalcemia was not reported.

**Adrenals.** Paired adrenals are found medial to and at the anterior end of the kidneys in both Asian and African elephants. They are "long, narrow and band like with a lateral horn" in African elephants,[46] but are more brick-shaped in adult Asian elephants (personal observation). In an Asian elephant fetus the left adrenal was said to be smaller and pyramidal, 4 cm long × 1 × 1 cm and weighing 5 gm; the right was 5 cm × 1 × 1 and 8 gm.[28] In a large study of adrenal gland weights in culled African elephants, Baranga[1] found that right adrenals were generally larger than left and that adrenal weight increased with age. Combined weights ranged from 10.9 gm in a 1-year-old female to 397 gm in a 60-year-old female.

Adrenals were weighed and measured in several of the UCDVMTH cases. In a 28-year-old captive African bull, each adrenal measured 27 × 5 × 2.5 cm. In three adult female Asian elephants, the left adrenals were 21 × 6 × 3 cm, 20 × 8 × 4 cm, and 21 × 6 × 2.5 cm, and weighed 261, 194, and 164 grams, respectively. The right

adrenals were 24 × 5 × 3, 26.8 × 9.2 × 4, and 26 × 7.5 × 3.0 cm, and weighed 220, 305, and 175 gm, respectively. In another 58-year-old female, the left adrenal was 28 × 8 × 3 cm and the right was 23 × 8 × 2.5 cm, but weights were not taken; in a 37-year-old female only the left adrenal was weighed and was 167 gm. The adrenal capsule is thick and opaque white, the cortex in all elephants examined has been dusky yellowish tan, and the medulla has been red or dark brown. The cortico-medullary ratio varies from 2:1 to 3:1.

The histology of the adrenal gland of African elephants has been described.[20,34] The capsule contains connective tissue and smooth muscle and a population of cells termed *capsule cells* that contain either glycogen or lipid and are of uncertain significance. Otherwise the histology is similar to that in many ungulates, though O'Donoghue[35] was struck by the abundant lipid droplets in the cortices of the adrenals from two bull African elephants they examined. There seems to be a debate about whether there is a provisional cortex in elephants; some authors say it is present and persists until the 4th year of life.[21]

Clinical assessment of adrenal function requires measurement of hormones produced by the gland—i.e., corticosteroids or mineralocorticoids, and response to stimulatory hormones (ACTH). Identification of clinical signs and laboratory values consistent with adrenal dysfunction is also necessary. Examples would be hypercholesterolemia and elevated alkaline phosphatase in hyperadrenocorticism (Cushing's syndrome) and hyperkalemia in hypoadrenocorticism or adrenal insufficiency (Addison's disease).

Blood (serum) levels of cortisol and catecholamines have been investigated as a means for assessing stress during the culling of African elephants.[7,13,14] A fluorometric method was used for cortisol and a radiometric/enzymetric assay for total catecholamines. Values for cortisol ranged from 66 to 825 nmol/l in the 1979 study and 11.38 to 858 nmol/l in the study by Hattingh.[13] Higher levels were found in animals culled after being disturbed. Similar results were found for catecholamines, with a range of 271.5 ± 28.2 to 355.7 ± 68.7. Lower mean levels were found in the least-disturbed animals. In a more recent study, serum levels of cortisol measured by solid phase RIA were determined to be independent of reproductive cycle in both Asian and African elephants.[8] Mean values were 20.04 ± 7.83 (range 7.59–73.54) ng/ml in noncycling Asian elephants and 27.53 ± 5.92 (range 4.05–110.91) ng/ml in noncycling African elephants (note different units for the previous studies).

Noninvasive testing for levels of glucocorticoids in urine and feces, as an indicator of stress and to investigate the phenomenon of musth, has been investigated.[10,11] In their study, Ganswindt[11] found that urine, not feces, was the excretory product of choice for measuring cortisol levels because 82% of metabolites of radio-labeled cortisol were excreted in the urine. They, along with Stead,[42] demonstrated that ACTH challenge results in increased excretion in cortisol and metabolites. Stead's group concentrated on use of fecal cortisol metabolite analysis as a more practical method for field work and compared blood and fecal levels after ACTH stimulation using an enzyme immunoassay and an antibody to 11-oxoaetiocholanolone.

Fecal cortisol metabolite concentrations were used to investigate social and environmental stressors.[10] Higher levels were significantly correlated with dry season and lack of rainfall in the wet season. Levels were also highest in groups with the most individuals and were somewhat correlated with degree of parasitism, and the authors suggested that stress-related immune suppression might play a role.

Several adrenal lesions were seen in the UCDVMTH case series. Nodular cortical hyperplasia appears to be a common finding in captive adult female Asian elephants and may be appreciated grossly as multiple 0.2 to 1.0 cm yellowish tan foci within the cortex or bulging from the surface. This lesion was reported in four Asian females in our pathology files, ages 37, 39, 54, and 58 years. A red, slightly cystic medullary mass in a 46-year-old adult female Asian elephant proved histologically to be a pheochromocytoma, which was not recognized clinically. Vacuolation of cortical cells (lipidosis) is another common finding in both Asian and African elephant cases in our files. Whether this represents increased activity due to stress or a degenerative change is a subject of debate for all species.[39] Multifocal adrenal cortical mineralization was seen in another old adult female Asian. A congenital anomaly of supernumerary adrenal glands was reported by Mikota.[32] The clinical significance of all these lesions is uncertain. No reports were found of either hyper- or hypoadrenocorticism in elephants in the literature.

**Endocrine pancreas.** The elephant pancreas is found adjacent to the duodenum as in other ungulate species.[9] The endocrine cells are organized into islets, and are also scattered individually and in small clusters among the exocrine acini. In the UCDVMTH immunohistochemistry lab, we have shown that commercially available stains for insulin and glucagon are broadly cross-reactive. Using immunohistochemistry, Van Answegen[46] identified insulin-containing cells in the core of islets and glucagon- and somatostatin-containing cells at the periphery.

No clinical reports of diabetes or metabolic syndromes associated with endocrine pancreatic dysfunction were found in the literature.

Pancreatic lesions found in elephants necropsied at the VMTH were confined to the exocrine pancreas in adult Asian females. There were two cases of nodular hyperplasia of acinar tissue and one case of mild chronic mononuclear interstitial pancreatitis.

**Other APUD structures.** Studies of the digestive system of the African elephant have shown that enterochromaffin cells occur throughout the gastrointestinal track.[45]

## THE IMMUNE SYSTEM

There is a paucity of literature on the anatomic and functional aspects of the elephant immune system. Much research is needed.

### Gross and Histologic Anatomy

The morphologic and functional anatomy of the lymphoid system of elephants has been poorly documented in the literature. As for all mammals, it is assumed that the thymus is a primary lymphoid organ for maturation of T lymphocytes and that bone marrow is the site of maturation of B lymphocytes. Secondary lymphoid organs include the spleen, lymph nodes, and tonsils. Accessory lymphoid tissues include the mucosal associated lymphoid tissues (MALT) of the respiratory tract (bronchial associated lymphoid tissue, BALT) and gastrointestinal tract (gut associated lymphoid tissues, GALT).

**Thymus.** In both African and Indian elephants the thymus is reported to lie wholly in the anterior mediastinum/anterior pericardial sac without cervical lobes similar to those found in bovids. The time of maximal development and timing of regression are unknown. Variations in size and weight would be expected to be a function of body weight or gestational and postnatal age; however, these parameters have not been fully documented. In the fetus described by Mariappa[28] the thymus was bilobed with total dimensions of 15 cm long × 7.5 cm wide.

The absence of thymic tissue in the anterior mediastinum in near-term fetuses and nursing calves may be expected to signal potential problems with immune competence. Thymic hypoplasia, atrophy, and premature involution may be due to malnourishment, infections (especially viral infections), or "stress." In domestic species, some degree of thymic "regeneration" may be expected after the insult is removed.

One Asian and four African elephant calves have been necropsied at the VMTH. The only thymic lesion reported was hemorrhage and the presence of inclusion bodies in an Asian calf with disseminated endotheliotropic herpesvirus infection. It is uncertain at what age the thymus reaches maximal size or when involution begins and is complete.

**Lymph nodes.** Many ungulate species have two types of aggregated lymphoid tissue or lymph nodes associated with lymphatics and the hemal nodes associated with the blood vascular system. Most authors agree that hemal lymph nodes occur in elephants.[9,28] Morphologically, hemal lymph nodes are dark reddish brown, and, in other species of ungulates, are distributed along the great vessels, especially in the anterior portions of the body cavities.[28] Mariappa states that hemal nodes are more numerous than lymph nodes and often occur as clusters of small spherical structures along vessels, including the mesenteric blood vessels, the hemorrhoidal arteries on the lateral walls of the rectum, lingual vessels, carotid artery, and jugular veins. Mariappa also documented larger solitary hemal nodes along the liver, external iliac artery, and anterior mediastinum; above the subcutaneous thoracic artery, vein and nerve; deep to the tendon of the latissimus dorsi muscle; and along the thoracic aorta. Hemal nodes are recognized by their dark red or brown coloration; however, lymph nodes draining hemorrhage, or with hemorrhage or containing hemosiderin may resemble hemal nodes. There is probably no clinical relevance to the question of presence or absence of hemal lymph nodes in elephants.

The distribution of lymph nodes in elephants has not been well characterized. The following descriptions come from personal observations. Mapping of lymph nodes was made easier in cases in which reactive hyperplasia or lymphadenitis was present. It appears that elephants have a chain of cervical lymph nodes from the postauricular subcutis to the angle of the mandible, continuing along the neck to within the thoracic inlet. The most anterior-dorsal node is just posterior to the external ear canal and somewhat distant from the rest of the chain in the location of parotid lymph nodes in other ungulates.

There is a cluster of at least five axillary lymph nodes and a similar cluster of inguinal nodes, with a separate node located more dorsally in the flank region that probably corresponds to the prefemoral node of ungulates. Woodford reports that parotid, mandibular, and superficial cervical and prescapular lymph nodes are located similarly to other species. These authors state that a popliteal lymph node is not present.

Within the thoracic cavity there are tracheobronchial nodes at the bifurcation of the trachea, often small and embedded in abundant hilar fat. The jejunal, ileocecal, and colonic nodes are often difficult to identify because they are embedded in the mesenteric fat and, in the absence of enteritis, are inconspicuous. Cheeran[9] states that there are only a few lymph nodes in the mesentery.

In an Indian elephant fetus, the lymph nodes were reported to have a subcapsular sinus but no distinct cortex and medulla.[28] However, in juvenile and mature elephants examined by the author, nodal organization was similar to that in most mammals.

Reported lymph node lesions include the common incidental occurrence of pyogranulomatous lymphadenitis in wild African elephants due to *Staphylococcus* sp., *Cryptococcus* sp., and other pathogens.[46] In captive Indian elephants, any lymphadenopathy associated

with caseous or granulomatous inflammation should be considered to be mycobacteriosis until proven otherwise by histopathology or molecular techniques.[22] Caseous lymphadenitis with mineralization associated with *Corynebacterium* sp. was seen in a 54-year-old female Asian elephant necropsied at the UCDVMTH. The most common lesion noted in lymph nodes in the UCDVMTH cases was reactive hyperplasia and drainage reaction.

Elephant lymph nodes, similar to those in bovids, are capable of undergoing remarkable lymphoid hyperplasia in response to chronic infections and inflammatory processes, with individual axillary nodes up to 18.5 × 10 × 5 cm noted in an Asian elephant with pododermatitis. In spite of this, given the location of peripheral nodes and the thickness of the hide, it is unlikely that lymph nodes can be palpated clinically.

Lymph node fibrosis is common in megavertebrates and elephants are no exception. This may result from chronic drainage reaction or lymph stasis due to inactivity.

Lymphosarcoma or other lympho-reticular neoplasms have not been described in elephants, although herpesvirus-associated pulmonary lymphoid nodules have been seen.[29] Lymph node metastases from other tumors are reported.[27]

**Tonsils.** Cheeran[9] states that tonsils are absent in elephants. Mariappa[28] described an obliquely oriented, 0.5 cm long by 0.5 cm deep tonsilar sinus in fetal Indian elephants located at the junction of the oral and pharyngeal palate, but tonsils were absent. However, a recent necropsy dissection of an adult female Indian elephant revealed well-developed discoid to spherical palatine tonsils on either side of the pharynx. The mucosal openings were a series of small pits that in turn opened into a submucosal cavity containing mucus. In an adult female the tonsil diameter was 5 cm. Histologically, the epithelium of the central cavity was evaginated into numerous crypts intimately associated with lymphoid tissue in the form of subepithelial follicles and numerous intraepithelial lymphocytes. Pharyngeal tonsilar tissue was also noted histologically in an adult female African elephant euthanized because of dystocia and toxemia. Thus it seems that pharyngeal tonsils do exist in both Asian and African elephants. The view to the contrary espoused in the literature is likely an artifact of the fact that much of the anatomy of elephants has been gathered from dissection of fetuses or neonates.

Based on examination of necropsy material from one Asian elephant, MALT is also associated with the palatine pits at the junction of the nasal passages of the trunk and the hard palate.

Lesions identified in the tonsilar tissue include crypt epithelial necrosis with neutrophil infiltration. In an adult Asian elephant, *Streptococcus agalactia* and *Candida* sp. were cultured. These lesions were likely subclinical.

**Spleen.** The spleen is an elongated flat, rectangular to crescentic organ on the left side of the abdominal cavity, between the stomach and body wall along the "left anterolateral aspect of the stomach."[9,46] The capsule is thick and opaque. The ligamentous structures associated with the spleen have been described as "gastrosplenic" and "mesoduodenum."

In an Asian elephant fetus, the spleen was 25 × 5 cm and weighed 107 gm.[28] The splenic weight of an African bull from the UCDVMTH files was 7.5 kg.

In free-ranging African elephants, subcapsular hemorrhages that elevate the contour are considered to be within normal limits.[46] The latter authors also note the finding of accessory spleens attached to the stomach in the gastrosplenic ligament.

Capsular siderofibrosis and red pulp hemosiderosis, either diffuse or nodular, are frequent findings in the spleens of adult elephants at necropsy. The former lesion suggests splenic trauma and hemorrhage, and the latter suggests either episodes of hemolysis or sequestration of iron secondary to chronic infections.

**Bone marrow.** There is little information in the literature on the distribution or histology of the hematopoietic marrow of elephants. See Chapter 25.

**Mucosal associated lymphoid tissue (MALT).** As described above, MALT is present in the form of palatine tonsils.

Intestinal MALT (GALT), in the form of individual glands and Peyer's patches, are described only in the duodenum by Mariappa.[28] In cases at the VMTH, small lymphoid nodules were widely scattered in the cecum and large colon of an Asian elephant, similar to the GALT seen in horses.

It is uncertain how much BALT should be present in the absence of disease. In African elephants, a herpesvirus associated nodular lymphoid hyperplasia associated with bronchioles has been described.[29]

## Assessment of the Immune System and Serologic Testing

Serum immunoglobulin and white blood cell counts, especially lymphocyte counts, are the only way clinically to assess elephant lymphoid system function antemortem, but the clinical pathologic correlates of changes in these parameters have not been well documented. Elephant lymphocyte subsets and immunoglobulins have been incompletely studied, though comparative use of immunohistochemical reagents in the UCDVMTH pathology service supports the presence of both B and T cells. In the absence of specific markers for identification of lymphocyte cell types, Rajan[37] used acid Alpha Naphthyl Acetate Esterase activity as a T cell marker, to demonstrate T cells in circulation. Also lacking in the literature is a detailed evaluation of acute phase proteins or lymphokines as early indicators of inflammation.

In African elephants, IgG appears to be the dominant immunoglobulin and at least 5 subclasses were identified by electrophoresis, SDS-page, and comparison with reagents reactive against immunoglobulins from other species.[18] The investigators were unable to confirm either IgM or IgA in the serum of adult African elephants, though this does not preclude the presence of these classes of immunoglobulins.

The Fc portion of African elephant immunoglobulin is reported to react with *Staphylococcus* proteins A and G, and these proteins have been used as linking molecules in ELISA-based serodiagnostics or in immunoprecipitation tests in the absence of a species-specific antielephant immunoglobulin.[2,18,31,43] Kelly[18] showed that several antispecies immunoglobulins reacted with elephant IgG obtained by ion-exchange chromatography of ammonium sulphate precipitate of pooled Africa elephant sera. Strongest reactivity was to polyclonal rabbit antibovine, dog, and camel. Strong reactivity was also seen with one mouse monoclonal antibody to human IgG. The authors suggested that these antibodies might be used for serodiagnostics, but this was not proven. Specific antispecies antibodies were developed in the course of studies on elephant endotheliotropic herpesvirus.[17]

Serologic surveys of either African or Asian elephants for agents as diverse as African horse sickness virus, poxviruses, plague bacillus (*Yersinia pestis*), and *Toxoplasma gondii* have used serum neutralization,[3,5] hemagglutination,[4,12,38] or modified agglutination test (MAT)[44] to obviate the issue of antispecies antibodies.

## CONCLUSIONS

Both organ systems covered in this chapter have been incompletely studied in elephants. Especially needed are reagents to better assess immunity in both Asian and Africa elephants.

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# 24 Cardiovascular System

Susan Bartlett

## INTRODUCTION

### Anatomy and Physiology

The elephant cardiovascular system has several unique characteristics. Although the typical mammalian heart is about 0.6% of the animal's body weight,[22] it is only about 0.5% of the elephant's body weight.[26] Depending on the elephant's size and age, the heart may weigh 12–21 kg.[25] The heart is often described as having a bifid apex, though this shape may be subtle or undetectable in elephants with a fatty mantle surrounding the heart (Fig. 24.1). The long axis of the heart is directed ventrocaudally. The ventricles lack trabeculae septomarginalis or moderator bands[15] and no *os cordis* is present.[26]

The pattern of vessels exiting the heart may vary. The right and left coronary vessels may originate from a single common branch or have two separate origins. In most animals the aorta gives rise to the left subclavian artery and the brachiocephalic trunk, from which arise the right subclavian and carotid arteries.[15] However, investigators have occasionally found the left common carotid, the left brachial artery, left subclavian, right subclavian, and a common trunk for the carotids originating from the aortic arch.[26] As in humans, the ductus arteriosus connects the left pulmonary artery to the aortic arch.

Paired cranial and a single caudal vena cavae return blood to the heart. The left cranial vena cava is smaller than the right, which receives blood from the azygous vein.[15] The ventral wall of the caudal vena cava is much thicker (10–20 mm) than the dorsal wall (~2 mm). The tunica media is minimal and often incorporated into the thickened, muscular tunica adventitia. The proposed function of the thickened ventral wall is to prevent the vessel from collapsing due to a sudden increase in intraabdominal pressure.[4] Also present in the root of the caudal vena cava is cardiac muscle, which may assist with venous blood return. Atrial granules are found in myocytes of the caudal vena cava; thus it acts as an endocrine organ secreting atrial natriuretic polypeptide.[6]

Arteriovenous anastomoses occur in the elephant. A prominent one is located between the temporalis muscle and the temporal gland where the superficial temporal artery and vein anastomose. In other mammals these anastomoses are thought to contribute to regulation of blood pressure, chemoreception, and heat dissipation.[1] Several venous plexuses also occur, including the tibial venous plexus behind the stifle joint and the pectoral venous plexus on the deep face of the posterior deep pectoral muscle.[15]

The elephant lacks an external saphenous vein, but an internal saphenous vein is present on the medial aspect of the pelvic limb and serves as a potential site for blood collection. Although the vein can be visualized through the skin, it is quite deep, and the needle must be inserted perpendicular to the leg. Other sites of vascular access include the cephalic vein on the proximal foreleg, the vena caudalis centralis, and the auricular veins on the caudal aspect of the ears. The vena caudalis centralis is found on the dorsal midline of the tail near the 14th caudal vertebrae (located where the caudal skin fold ends on the ventral aspect of the tail). This vein may be reached with an 18-gauge needle directed ventrocranially inserted about 8 mm deep.[19] The auricular veins are easily accessed for venipuncture, but care must be taken when giving intravenous injections through these veins because sloughing may occur with extravasation of drug into the subcutaneous tissue.

Young elephants may be auscultated with a conventional stethoscope but the heart is difficult or impossible to hear in adults. Digital, electronic stethoscopes that magnify sound may be helpful, although interfering sounds may also be amplified. The pulse may be evaluated by manual palpation of the auricular (Fig. 24.2) or caudal vertebral artery. The latter site, on the ventral aspect of the tail, may be challenging because elephants commonly swing their tails when people attempt to restrain them.

Physiological ranges for cardiovascular assessment of

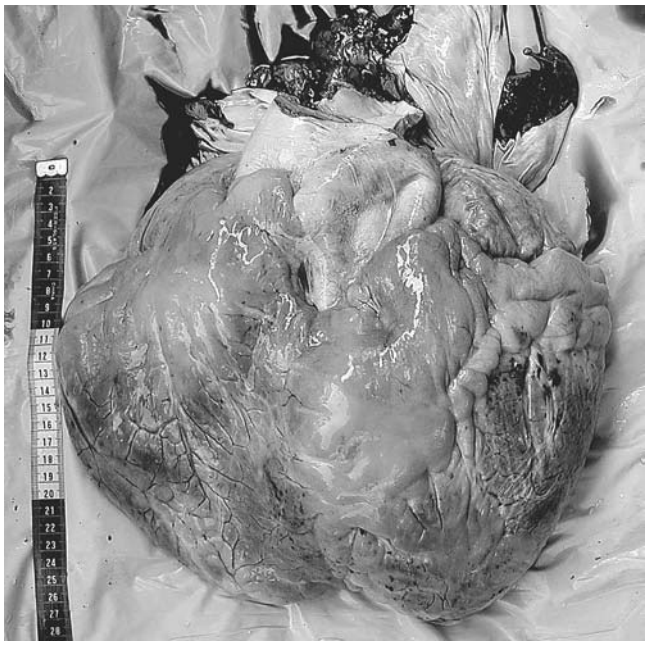

***Figure 24.1.*** *Elephant heart. The bifid cardiac apex is an anatomic feature typical of elephants and also of aquatic mammals in the order Sirenia (photo courtesy of Hank Hammatt).*

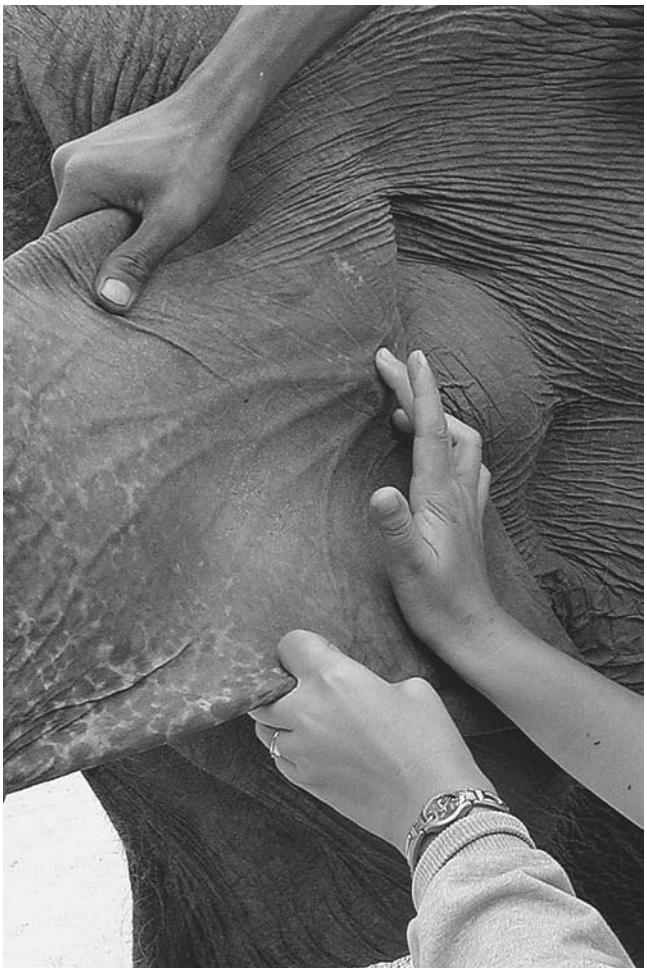

***Figure 24.2.*** *Asian elephant ear showing site for palpation of pulse and auricular veins, commonly used for venipuncture (photo courtesy of Hank Hammatt).*

standing African and Asian elephants are as follows: heart rate 24–50 beats per minute (bpm), averaging around 35 bpm,[23] mean arterial blood pressure 144.6 ± 2.9 mm Hg, systolic pressure 178.6 ± 2.9 mm Hg, and diastolic pressure 118.7 ± 3.1 mm Hg. These parameters increase when the elephant is in lateral recumbency. In one study the blood pressure of an elephant in lateral recumbency for 16 minutes averaged 179.8 ± 9.3 mm Hg.[9] The blood volume of one adult Asian elephant was approximately 3.5% of the animal's body weight.[25]

## ELECTROCARDIOGRAPHY

Electrocardiography of elephants was performed as early as 1921. Several attempts have been made since that time with increasing success. However, the challenges facing early investigators are the same ones veterinarians face today: selecting an appropriate electrode, maximizing conduction of the signal from the elephant's skin to the electrode, and minimizing artifact due to movement.

In 1938 a galvanometer was used to obtain electrocardiograms (ECGs) on nine elephants.[30] Electrodes consisted of silver plates covered with cloth soaked in concentrated salt solution. The elephants either stood on the electrodes or they were strapped to the feet when lying down. The complexes were well defined but of low amplitude. Jayasinghe attempted ECGs on three occasions. The first was with an adult Asian female using nickel-silver electrodes and gel with standard limb leads.[12] The poor contact of the electrodes with the skin impaired the quality of the readings. During his second and third attempts—on a 1-year-old Asian elephant and two adult Asian females, respectively—he used alligator clips with electrode gel, using both limb and chest leads with improved results.[10,11]

In 1967 Geddes[8] obtained ECGs with novel electrodes designed to minimize artifact from animal motion. The electrode consisted of a shallow cup with a silver metal disc mounted in the center. The cup was filled with electrode gel and held in place with rubber straps. Because the electrode metal did not directly contact the skin, motion artifact was minimized. She used limb leads and a chest lead and obtained good waveforms.[8] In 2002 the author obtained ECGs from three Asian female elephants, two adults and one juvenile, using another novel clamp. This clamp was a modified 2-inch hand vise with a copper plate replacing one of the clamp's plastic end plates. Limb leads, chest leads, and a base-apex were obtained with good waveforms (Bartlett, unpublished).

**Table 24.1.** Duration of Elephant ECG Waves and Intervals in Seconds

| Investigator n = # of Elephants | Heart Rate (b/m) | P Range (avg) | P–R Range (avg) | QRS Range (avg) | T Range (avg) | Q-T Range (avg) | Q–U Range (avg) |
|---|---|---|---|---|---|---|---|
| White[30] n = 9 | 24–53 | 0.12–0.20 (0.16) | 0.28–0.41 (0.36–0.37) | 0.12–0.18 (0.16) | — | 0.59–0.79 (0.65) | — |
| Jayasinghe[11] n = 2 | 32–48 | 0.12–0.20 (0.16) | 0.24–0.48 (0.36) | 0.06–0.16 (0.14) | 0.20–0.28 (0.24) | 0.58–0.68 | — |
| Jayasinghe[10] n = 1 | 37 | 0.08–0.10 (0.08) | 0.28–0.32 (0.28) | 0.08–0.10 (0.08) | 0.32–0.44 (0.40) | 0.60–0.64 | — |
| Geddes[8] n = 4 | 28–50[a] | — | 0.37–0.45 (0.39) | 0.07–0.16 (0.10) | — | 0.55–1.00 (0.67) | 0.80–1.38 (1.08) |
| Loypetjra[14] n = 4 | 35–49 | 0.04–0.08 | 0.28–0.36 | 0.04–0.08 | — | 0.60–0.72 | — |
| Sreekumar[29] n = 1 | 31–46 | 0.16 | 0.26–0.38 | 0.25–0.32 | — | — | — |
| Mikota[20] n = 1 | (60)[b] | (0.16) | (0.32) | (0.16) | — | (0.66) | — |

a) the elephant with a heart rate of 50 was exercised
b) recorded in lateral recumbency

**Table 24.2.** Amplitude of Elephant ECG Waves in Millivolts

| Investigator n = # of Elephants | Heart Rate (b/m) | Lead | P Range (avg) | Q (avg) | R (avg) | S (avg) | QRS Range (avg) | T Range (avg) |
|---|---|---|---|---|---|---|---|---|
| White[30] n = 9 | 24–53 | I | 0.05–0.15 (0.1) | — | (0.6–0.8) | (0.1–0.2) | — | 0.05–0.20 |
| Jayasinghe[10] n = 1 | 37 | — | (0.1) | — | — | — | — | — |
| Loypetjra[14] n = 4 | 35–49 | II | 0.05–0.10 | — | — | — | 0.1–0.6 | 0.075–0.3 |
| Mikota[20] n = 1 | 60 | II | (0.075) | (0.04) | — | (0.0) | — | (0.15) |

A summary of the waveform measurements for selected studies can be found in Tables 24.1 and 24.2. Most investigators find that when using the bipolar limb leads alone or the hexaxial lead system, Lead I captures the clearest waveforms. Almost invariably investigators note low amplitudes of the complexes, especially those of the P and T waves. Possible explanations include the long distance between the heart and the electrodes, especially when they are attached to the distal limbs, the thickness of the skin, the types of electrodes used, and the types of recording apparatus.[10] Jayasinghe noted large and clear waveforms when recording an ECG from a 1-year-old elephant, and attributed this to the thin skin. To maximize the clarity of the waveforms the author recommends amplifying the waves when recording ECGs. Paper speed may be set to 25 or 50 mm/s.

Typical elephant ECGs consist of P, QRS, T, and U waves. The T waves, representing repolarization of the ventricles, may be low, domed, or sagging. The U waves in certain recordings may be subtle and difficult to identify. Geddes[8] suspects the U waves are due to an overshoot in the repolarization of the ventricles or a delayed repolarization in some areas of the ventricles. White[30] noted that the long durations of the P–R and Q–T intervals and the long QRS wave width are likely due to the size of the elephant heart. The Q–T interval, which is a measurement of the duration of systole, decreases as heart rate increases.[11,30] The S–T segment does not normally deviate from the baseline.

Position of the animal does not appear to affect the ECG greatly. Although in lateral recumbency the heart rate tends to increase, with a concurrent shortening of the Q–T interval, the duration of the waveforms is not significantly affected. White noted that recumbency led to decreased amplitude of the P and T waves, and to a lesser extent the QRS complex, attributing this to a change in heart position relative to the cables.[30] The estimated mean electrical axis in a standing juvenile elephant was 40–90°.[14]

ECGs may be useful in assessing the cardiovascular health of elephants. Mean resting heart rate and the diastole-systole quotient (DSQ) were used as measurements of cardiac fitness in a study comparing relatively sedentary zoo elephants with circus elephants that trained in a fitness program prior to touring.[21] The zoo elephants had high resting heart rates (48 bpm) and low DSQs (1.34), which were interpreted as poor cardiac fitness. Prior to the exercise program for the circus elephants, the resting heart rates were high (41 bpm) and the average DSQ was low (1.53). As the elephants trained over the course of 8 months, the resting heart rates decreased to a low of 29 bpm and the DSQ increased to 2.16, indicating improved cardiac fitness. Four weeks after training ended, the heart rate and DSQ nearly returned to their pretraining values. An ECG was also performed on a 6-year-old Asian elephant with signs of cardiac insufficiency (generalized weakness, increased respiratory rate, edema of all four legs, and lethargy).

The ECG showed a high heart rate (49 bpm) and a low DSQ (1.16). Mill also cited another study of an elephant with cardiac failure whose ECG showed a long QRS duration (0.21 sec) and a deviation of the S–T segment.[21]

The common options of electrodes available to veterinarians include alligator clips, hypodermic needles, and adhesive ECG patches. Alligator clips may be effective for limb leads, though the waveforms tend to be of low amplitude and will easily slip off the elephant's skin. They are very difficult to use for chest leads because there is minimal loose skin to grasp. Hypodermic needles may be inserted into the skin and alligator clips attached to the needles, thereby producing clearer waveforms, though puncturing the animal's skin is not ideal. ECG patches will not adhere well to the skin of the elephant, but if reinforced with duct tape or the like and if ample conducting gel is used, an adequate signal can be obtained (Fig. 24.3).

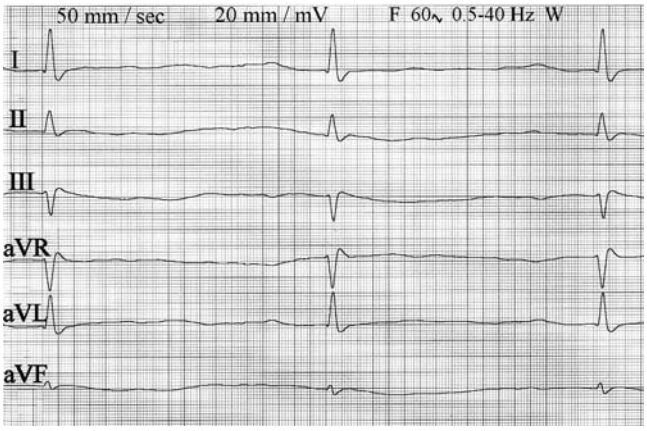


***Figure 24.3.*** *ECG tracing from a healthy 26-year-old female Asian elephant. The ECG leads were attached with adhesive patches.*

Motion artifact is another obstacle. Healthy elephants are almost always in motion. Trained elephants may be asked to perform a command such as lifting the trunk or a foot to decrease motion. However, the latter command is best performed for brief periods of time because muscles will tire and lead to tremors and artifact. When using a base-apex configuration, it is often necessary to restrain the elephant's ear manually, or else the flapping will jostle the lead. Long cables (~75 cm; 30 inches) are necessary to connect the electrodes and the ECG machine. As the elephant sways so will the cables; stabilizing them with a hand may help minimize artifact. To further minimize artifact, the author recommends turning on the filter.

## CARDIOVASCULAR DISEASE

Cardiovascular disease in captive elephants appears to be a relatively uncommon occurrence, though when it does occur it often has serious ramifications. In a survey of 379 captive elephants, only 19 were documented to have cardiovascular disease. However, 18 of these 19 elephants died, with 11 of the deaths directly related to their CV disease.[20] Arterial disease is commonly documented, especially in free-ranging elephants.

### Arterial Disease

In 1966–1967 McCullagh and colleagues examined the cardiovascular systems of 463 randomly culled free-ranging elephants in Kenya and Uganda.[18] They found lesions in 298 aortas (72%) and 29 coronary arteries (27%). In another study of 207 culled elephants in Zambia, over 60% were free of cardiovascular lesions.[5] Arterial disease occurs in two different forms: intimal atherosclerosis and sclerosis of the tunica media. Intimal atherosclerosis is characterized by discrete, white, firm, raised plaques measuring 1–50 mm in diameter usually found in the aorta, coronary, carotid, renal, and iliac arteries. The plaques are fibrous with histological evidence of degenerative changes (lipid accumulation, lymphocytic infiltration, calcification, hyalinization, and necrosis). Unlike the condition found in humans, no mural thrombosis or plaque hemorrhage is present. The etiology of the plaques has been debated. Some investigators (McCullagh[17] and Dillman[5]) believe the lesions arise as a result of repeated mechanical trauma to the endothelium from normal blood flow. They did not find any correlation between concentrations of serum cholesterol, phospholipids, triglyceride, or free fatty acids and the severity of disease. It has been postulated that the lipid accumulation in the lesions actually occurs after the fibrous plaque begins to form. McCullagh and Dillman found that the lesions increased in number and severity with increasing age; yet the severity of lesion did not appear to correlate with body condition.

Medial sclerosis is characterized by irregularly rounded, flattened plates 3–15 mm in diameter located primarily in the aorta, coronary arteries, and aortic branches. The process often begins in the abdominal aorta, especially at the quadrifurcation, and proceeds anteriorly into the thoracic aorta. The more calcified plates are hard, brittle, and golden-brown in color. Histology shows atrophy of medial smooth muscle fibers with secondary fibrosis and calcification of the internal elastic lamina. The endothelium covering these plates is usually normal, though concomitant intimal atherosclerosis may be seen. McCullagh[16] found a positive correlation of the progression and severity of the medial sclerosis with increasing age and higher serum calcium concentrations. The high serum calcium does not, however, initiate the lesion but rather exacerbates the process after the medial degeneration has begun. Dillman did not find any difference in the serum concentrations of sodium, calcium, and magnesium between affected and unaffected elephants, though the serum potassium concentration was lower in animals with lesions.[5] This decrease in potassium concentration

may simply be age related, however. Etiology of these lesions is speculative, but possible causes proposed by McCullagh include medial anoxia of large muscular arteries, which lack vasa vasorum and mechanical trauma from the repeated pulsations with secondary degeneration, fibrosis, and calcification. McCullagh did not find a significant difference in severity of disease between male and female elephants.

In 1964–1965 Sikes studied 40 free-ranging elephants in Kenya and Uganda. She observed elephants pre- and postmortem in natural (montane) and disturbed (grassland and scrubland) habitats and proposed that arterial disease arises in elephants due to living in disturbed environments.[26] The elephants living in the montane habitat did not have any evidence of arterial disease and had firm, long hearts. Elephants living in disturbed habitats had both types of lesions and tended to have a square, flaccid heart with a more prominent bifid apex. Elephants with aortic lipidosis did not show premortem clinical signs. However, those with advanced medial sclerosis showed clinical signs, including general emaciation, immobility, sluggishness, and drooped head and ears, which Sikes speculated was due to compromised blood flow to the head, heart, and limbs.[27] She found elephants with partially occluded iliac and femoral arteries, including one elephant that had secondary vascular compromise with a swollen foot, varicose veins, and a soft sole. This contrasts with the findings of Dillman, who did not observe any loss of body condition even in those with the heaviest arteriosclerotic lesions.[5] Sikes did not believe either disease was directly related to age, but rather that imbalanced habitats led to nutritional deficiencies, increased sun exposure with the potential for hypervitaminosis D, and overpopulation.[28] She did note, however, that a pointed arch of fibrous supportive tissue located at the bifurcation of the aorta is prone to an age-related mineralization.

Arteriosclerosis has also been documented in captive elephants and at times has been fatal. Lindsay describes the necropsy findings of a female Asian elephant at least 47 years old that died acutely of myocardial failure due to severely compromised coronary blood flow. Postmortem findings included pericardial effusion, intimal atherosclerosis and medial sclerosis of both coronary arteries, and narrowed lumens of the majority of small coronary branches of the left ventricle.[13] Although the morphological pattern of arterial disease is similar for captive and free-ranging animals, conditions in captivity may potentiate lesions.[7]

## Cardiomyopathy

Cardiomyopathy has been documented in several captive elephants. A female African elephant that died at age 44 had a history of subtle though progressive signs of disease, beginning at age 36. At this time she experienced episodes of ventral edema, followed by development of a mild anemia that persisted until her death. At 42 years of age she subjectively appeared to begin losing weight and began spending more time resting in a stationary position with or without concurrently resting her head against a tree post. Also noted at this time were daily episodes of skin twitching in her chest. As these signs progressed over the course of a year, she also became increasingly lethargic and nonresponsive. The elephant was found in sternal recumbency one morning unable to rise and died despite supportive care. Necropsy findings included cardiomyopathy with marked perivascular and dissecting fibrosis, moderate myofiber atrophy, myofiber anisokaryosis and karyomegaly, and myofiber lipofuscinosis. The ventricles showed moderate epicardial fibrosis and steatosis (replacement of muscle fibers with adipose tissue cells). In addition the elephant had moderate arteriosclerosis, which may have contributed to cardiovascular collapse following her prolonged recumbency (personal communication, Dr. Freeland Dunker, San Francisco, California, June 2005).

Hypertrophic cardiomyopathy was documented in a female African elephant (approximately 20 years of age) that died shortly after transport. The elephant was recumbent upon arrival at the receiving institution and did not respond to supportive care (vitamin E, selenium, flunixin meglumine, antibiotics, and intravenous fluids). Even when supported with a sling she could not stand and eventually became agonal and died. Premortem bloodwork indicated polycythemia, hypernatremia, hypercreatinemia and myopathy: LDH 6870 IU/L, AST 972 IU/L, and CK 69,938 IU/L. Histopathology findings included numerous enlarged nuclei in the myocardium with associated hypertrophic fibers and splenic hemosiderosis (personal communication, Dr. Kirk Suedmeyer, Kansas City, Missouri, June 2005).

## Viral

Two main viral infections cause CV disease in elephants. Encephalomyocarditis virus, a member of the *Picornaviridae* with a worldwide distribution, causes acute fatalities in both captive and free-ranging elephants. Vague clinical signs of anorexia, listlessness, and moderate dyspnea last about 24 hours before death, which is usually attributed to myocarditis with resultant cardiac failure. Disease caused by elephant endotheliotropic herpesvirus (EEHV) also presents acutely and may be fatal. EEHV disrupts endothelial cells in various organs including the heart, resulting in intramyocardial hemorrhage and edema with resultant cardiac failure. These are discussed further in Chapter 11.

West Nile virus (WNV) also has the potential to cause cardiovascular disease in elephants. Two captive adult African elephants that died of cardiovascular disease had recently been exposed to and possibly infected with WNV. One elephant, a 53-year-old bull, demonstrated a positive titer to WNV in May 2003. The titer continued to rise from 1:64 to 1:128 through June, during which

time the bull developed clinical signs including inappetance, lethargy, an unwillingness to respond to commands, Parkinsonian-like tremors of the trunk, and slightly loose stool. Laboratory evaluation revealed an initial leukopenia ($<5.0 \times 10^3$/ul), a beta globulinemia on serum protein electrophoresis, and a mild hyperfibrinogenemia. This was followed by a rebound leukocytosis, and then normocytosis. The bull responded well to antiinflammatory treatment (flunixin meglumine) and the WNV titer returned to baseline by July 2, 2003. The bull appeared normal clinically and hematology values returned to normal by the beginning of July. The bull died in September 2003 of cardiac degeneration and myocardial necrosis of unknown etiology.

The other elephant, a 26-year-old bull, demonstrated similar clinical signs and changes in laboratory values associated with a rising titer to WNV that exceeded 1:8000. Although he also appeared to recover, he died 4 weeks after the initial signs were noted. Premortem signs included a 72-hour progression of ataxia and hyperesthesia with eventual recumbency, though prehension and appetite were normal during this time. The WNV titer was negative on the day of death and endocarditis was diagnosed at necropsy. Both bulls tested negative for EMCV, and no WNV was found in the tissues of either animal (personal communication, Dr. Kirk Suedmeyer, Kansas City, Missouri, June 2005). Refer to Chapter 11 for more information on viral diseases.

### Other Conditions

Other conditions known to affect the CV system of elephants include invasion of vessel walls by migrating parasites, such as *Dipetalonema gossi*, with the potential weakening of vessel walls leading to aneurysm.[2] See Chapters 12 and 24. One case of tetrology of Fallot[24] and two cases of patent ductus arteriosus (PDA) have been documented postmortem in neonatal elephants, though the PDAs may have been physiological. Death due to cardiogenic shock secondary to *Eschericia coli* septicemia has been reported,[20] as well as an instance of suspected cardiac glycoside toxicity in two young wild African elephants that consumed a toxic plant.[3]

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# 25 Hemolymphatic System

Susan K. Mikota

## GLOSSARY OF TERMS

**CBC:** complete blood count

**Hct (hematocrit):** the volume percentage of erythrocytes in whole blood determined by centrifuging blood to separate cells from plasma; the ratio of cell volume to plasma volume is the packed cell volume (PCV)

**Hb (hemoglobin):** protein in erythrocytes that transports oxygen

**ESR (erythrocyte sedimentation rate):** a measure of the extent of settling of RBCs in a column of blood per unit time

**MCH (mean corpuscular hemoglobin):** the average Hb content of a single cell; MCH (picograms) = (Hb concentration × 10) ÷ RBC count (millions)

**MCHC (mean corpuscular hemoglobin concentration):** the average percentage hemoglobin concentration: MCHC (g/dl) = (Hb concentration × 100) ÷ Hct

**MCV (mean corpuscular volume):** an expression of the average volume of individual RBCs in cubic microns, calculated as follows: MCV (fl) = (Hct × 10) ÷ RBC count (millions)

**PCV (packed cell volume)**: the percentage of erythrocytes in whole unclotted blood

**RBC:** red blood cell; erythrocyte

**WBC:** white blood cell; leukocyte

## OVERVIEW OF THE HEMOLYMPHATIC SYSTEM

The primary components of the hemolymphatic system discussed here are 1) the bone marrow and the cells that it produces, 2) the thymus, 3) the spleen, 4) the lymph nodes, and 5) the lymphatic vessels.

Pluripotential stem cells in the bone marrow give rise to multipotential myeloid and lymphoid stem cells. Lymphoid stem cells differentiate into T and B lymphocytes. Myeloid stem cells differentiate into precursor cells that mature into the cell lines comprised of erythrocytes, neutrophils, monocytes, platelets, eosinophils, and basophils. The bone marrow is actively hemopoietic in early postnatal life in all species, but with maturity it recedes to the cancellous ends of the long bones, the vertebrae, and the flat bones (e.g., ribs). The red, hemopoietically active marrow is replaced by yellow, fatty marrow; the degree to which this occurs varies between species. Hemopoietically active areas (and sites for bone marrow biopsy) in adult mammals include the iliac crest (humans, dogs, horses), the sternum (cows, horses) the ribs (horses) and the vertebral process (cows). Bone marrow is lacking in the long bones and ribs of elephants[73] but may be collected from the dorsal spinous process of the first lumbar vertebrae.[11] General indications for bone marrow examination include persistent neutropenia, unexplained thrombocytopenia, osteomyelitis, or suspicion of hematopoietic neoplasia or infiltrative or proliferative bone marrow disease.[29]

Lymphocytes originating in the bone marrow undergo development into T cells in the cortex of the thymus, a lymphoid organ necessary for normal immunologic development. In most species, it is maximally developed at puberty after which it slowly involutes. The thymus is located in the anterior mediastinum and occupies the space between the thoracic inlet and the heart. In the elephant, the thymus has a pyramidal shape and is bilobed.[48]

The spleen is a large vascular lymphatic organ located on the left side of the abdomen between the stomach and the diaphragm. Although it is an active erythropoietic site in the fetus, it functions primarily as a filter in the adult. The spleen and thymus have efferent but not afferent lymphatics; therefore, antigens entering these organs do so through the blood vascular system.[94] Species that are "athletically active" and likely to flee in the face of adversity (humans, dogs, cats, horses) possess spleens with a contractile muscular capsule. In contrast, ruminantlike species that form groups to ward off predators have spleens with capsules that are mostly connec-

tive tissue.[94] The architecture of the spleen (and the predator response) of the elephant resembles the latter group and thus excitement-induced splenic contraction is unlikely to occur in the elephant.

The lymphatic system provides an essential route whereby proteins and particulate matter too large to be absorbed by capillaries are removed from the interstitial space and returned to the blood. Almost all tissues of the body have lymphatic channels that absorb excess interstitial fluid from which lymph is derived. About 10% of the blood that passes into the tissues from arterial capillaries returns through lymphatic rather than venous channels.

In the elephant, lymphatic vessels from the mesentery, stomach, and lumbar areas form trunks that pass through the diaphragm and coalesce into the cisterna chyli from which the thoracic duct arises. The thoracic duct empties into the left anterior caval vein[48] and drains the caudal body.

The lymphatic system in the elephant is said to be poorly developed, occupying only the splanchnic area, with the possibility that lymph may drain directly into the venous system in other locations.[48] Small, grey lymph nodes are found in the mesentery. Hemal nodes, which are dark brown, are more numerous and there are two types. Small round hemal nodes occur in groups or chains and are associated with the mesenteric, hemorrhoidal, and lingual blood vessels. A chain of eight, small, elongated nodes are found along the carotid sheath and jugular vein. Large hemal nodes are found in proximity to the portal fissure, thymus, pericardium, and aorta.[48] The cervical lymph nodes are also of the hemolymph type.[21] The interstitial lymphatics of the African elephant testis have been described.[31]

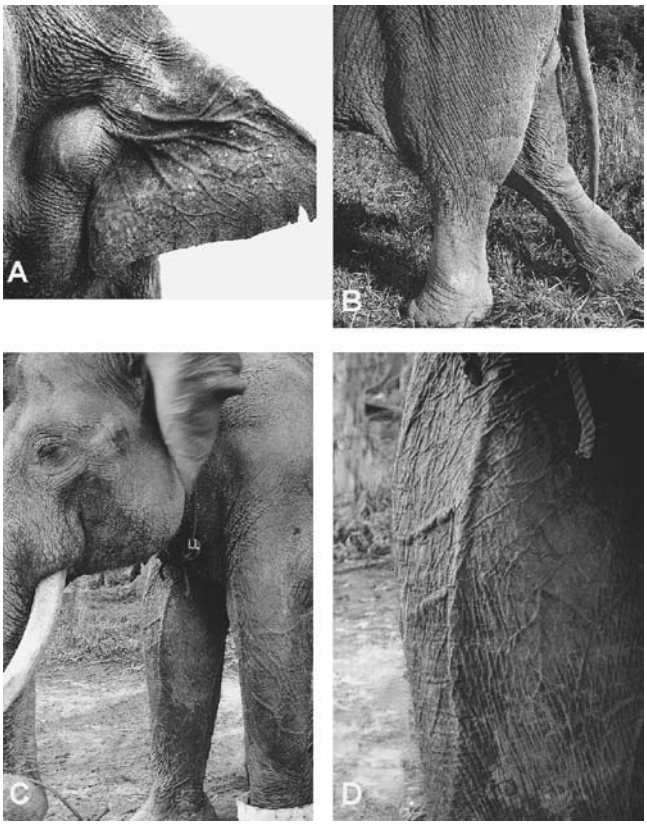


***Figure 25.1.*** *Bleeding sites. A) Auricular veins on the caudal pinnae of an Asian elephant, B) saphenous vein on medial aspect of hindleg (this vein is deeper than it appears, see text), C and D) cephalic vein on medial aspect of foreleg (photos A, C, and D courtesy of Hank Hammatt; photo B courtesy of Carol Buckley).*

## HEMATOLOGY

### Blood Collection Sites and Techniques

The auricular, cephalic, and saphenous veins are the primary blood collection sites for elephants[53] See Figure 25.1. Blood collection from the dorsal tail vein has been described,[99] but it is not commonly used. The auricular veins on the caudal aspect of the pinnae may be accessed with the elephant standing; however, the veins are more prominent and the elephant is less likely to move in lateral recumbency. In some elephants, these veins can also be visualized on the rostral aspect of the pinnae. Auricular veins may be distinguished from adjacent arteries by their thinner walls and absence of a pulse. The application of moist, warm compresses or dry heat (e.g., a hair dryer) will facilitate vasodilation when the ambient temperature is low. The auricular veins are involved in thermoregulation and can dilate and constrict dramatically.[19,74] The cephalic vein on the proximal medial forelimb is best visualized with the elephant standing; the saphenous vein on the lower medial aspect of the hindlimb can be seen with the elephant standing, stretched, or in lateral recumbency. The saphenous vein is surprisingly deep and it is usually necessary to position a 1 in needle at a 90° angle and insert it to its full depth in adult elephants. Winged blood collection sets tolerate a degree of movement without dislodging from the vein, permit collection directly into tubes, and are convenient for multitube collection. See Figure 25.2.

### Erythrocyte (RBC) Morphology and Indices

The elephant RBC is a nonnucleated biconcave disc. It is the largest mammalian RBC with a mean diameter of >9 µm,[35,47,66] a mean corpuscular volume (MCV) of 81–160 femtoliters (fl),[75,76] and a hemoglobin concentration of 7.4–15.8 pg.[73,74] The RBC count is lower than in other mammals[59] because of the large cell size. The life span of the elephant RBC has not been reported, but it is probably comparable to the 160-day longevity seen in the horse.[29]

Elephant RBCs have a high resistance to osmotic lysis, which may be related to the large surface/volume ratio of the biconcave disc. This may be of functional importance in preventing RBC lysis in an elephant that

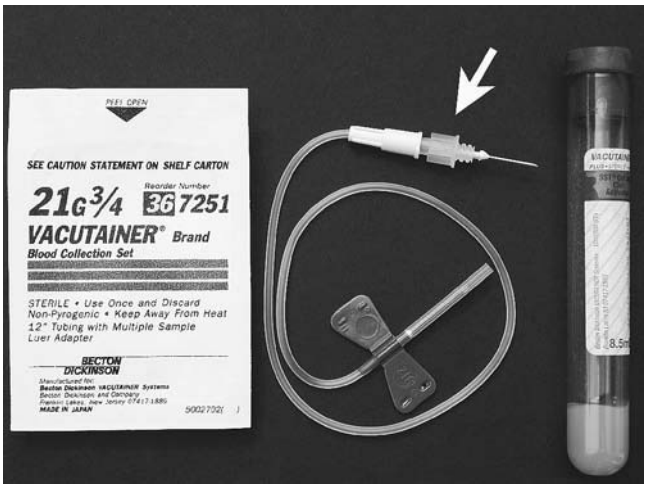


***Figure 25.2.*** *Winged blood collection sets (Vacutainer®) are convenient for obtaining multiple samples and are available in 21, 23, and 25 ga × 3/4 inch sizes. The adapter (arrow) may be purchased separately (Vacutainer®) and used with larger, 19 ga winged (butterfly) sets, the author's preference for adult elephants.*

**Table 25.1.** Red Blood Cell (RBC) Reference Values[a]

| Parameter | Units | Reference Range |
|---|---|---|
| PCV | % | 30–40 |
| Hb | g/dl | 11–15 |
| RBC | x $10^6$/µl | 2.5–5.0 |
| MCV | fl | 80–160 |
| MCH | pg | 35–50 |
| MCHC | g/dl | 25–40 |
| Platelets | $10^3$/µl | 200–600 |
| Reticulocytes | % | 0.00 |
| RDW | % | 28–32 |
| ESR | mm/hr | 65–150 |

[a]References: Silva 1993a, b; Niemuller 1990; Ratnasooriya 1990; Sreekumar 1990a; Jainudeen 1971; Nirmalin1971; ISIS 2002.

**General Table Disclaimer:** The reference values suggested in the tables in this chapter are derived from reported studies, with preference given to data from clinically healthy elephants.

Individual sources are cited where appropriate. These suggested ranges are to be used as a general guideline rather than as specific cutoffs to define normal versus abnormal. Laboratory values are affected by numerous factors (see text), and they may vary between individual elephants. Establishing a baseline hematological and serum chemistry profile for each elephant during health is recommended.

consumes a large volume of water following severe dehydration or thirst.[77]

Rouleaux formation, in which erythrocytes resemble stacked coins, occurs,[75,85,100] although it has not been detected in some studies.[41,66] Rouleaux formation is common in healthy horses but may be absent in anemic or emaciated animals.[29] Rouleaux formation in elephants may partly explain the high ESR, which is normal for the species (65–150 mm/hr). This characteristic is beneficial in separating out the serum of elephants in the field (personal communication, Dr. Indira Silva, Sri Lanka, July 2005).

Reticulocytes have not been reported in the literature, but they have been observed in anemic Asian elephants (personal communication, Dr. Indira Silva, Sri Lanka, July 2005). Reticulocytes do not occur in horses.

Erythrocyte parameters will change with increases or decreases in the circulating RBC mass. A decreased RBC mass (anemia) will be associated with decreases in the PCV, hemoglobin (Hb), and RBC count. The decreases may be disproportionate if there are concurrent changes in cell size and/or Hb content. An increased RBC mass (polycythemia) will elevate PCV, Hb, and RBC values. The red cell distribution width (RDW), measured by some automated counters, is a measure of the degree of anisocytosis. The RDW values will increase with significant microcytosis, macrocytosis, or reticulocytosis in domestic species.[29] It is not known, however, whether this is a useful parameter for elephants. Erythrocyte parameters are used to evaluate anemia (discussed below).

Physiological status should be considered when interpreting the hemogram. Dehydration may cause spuriously high values; overhydration may lower values, mimicking anemia.[29] From a clinical standpoint, hemoglobin concentration (Hb) is the best indicator of the oxygen-carrying capacity of the blood. If the RBCs are normal in size, the Hb should be approximately one-third the value of the PCV. Lipemia may falsely elevate Hb.[29] See Table 25.1 for suggested reference values for elephant RBC parameters.

## Erythrocyte Sedimentation Rate (ESR)

The ESR is the rate at which erythrocytes sediment when anticoagulated blood is placed in a vertical column. It is generally expressed as the fall of RBCs in mm at the end of 1 hour. The elephant has the fastest ESR of any mammalian species evaluated, attributable to high fibrinogen and globulin levels, large RBC size, low RBC count, formation of rouleaux, and low plasma albumin levels.[59,85] Variations in ESR values reported for elephants may reflect differences in techniques or times of measurement. The Westergren technique has yielded both higher[76] and lower[66] values for ESR than the Wintrobe technique.[41,59,79] Measuring the ESR at 15 minutes appears to be more accurate in elephants.[85]

The ESR is a nonspecific indicator of disease and/or response to therapy. Increases in ESR are typically associated with inflammatory diseases or chronic immunological stimulation.[96] With a normally rapid ESR, the variation that may occur in disease states may not be clinically important in elephants; however, significantly higher ESR (and lower PCV and RBC values) have been reported in pregnant Asian elephants.[59]

## Physiological and Environmental Effects

In healthy elephants, the hematological profile is constant over time and similar between individuals.[32,58,66]

Values for African and Asian elephants are similar, and differences between genders or between captive and free-ranging Asian elephants are insignificant.[75,76] Pregnancy appears to have the most dramatic effect on hematological parameters; pregnant Asian elephants have lower PCV and RBC counts with a corresponding lower MCV and higher sedimentation rates than nonpregnant, nonlactating adult females.[59] This effect has not been noted in pregnant African elephants.[17] Habitat-induced cardiovascular disease has been implicated as causing variations in PCV and RBC counts in free-ranging elephants from two different habitats in Uganda.[97] See further discussion in Chapter 24.

Environmental factors have been shown to influence the platelet count (positive correlation with ambient temperature, rainfall, and day length), MCV and MCH (positive correlation with average temperature and day length), and RBC count (negative correlation with average temperature and day length) in Asian elephants.[64] Lower RBC and higher PCV values have been observed in the winter in captive Asian elephants.[35] Higher Hb, MCH, and MCHC values and lower MCV values in free-ranging African elephants in the dry season may result from dehydration.[97]

## Platelets and Coagulation

Blood coagulation is a dynamic system designed to prevent blood loss through fibrin formation following endothelial damage and to maintain blood flow by fibrinolysis following tissue repair. Primary hemostasis is the mechanism whereby platelets adhere to injured endothelium. Secondary hemostasis is the sequence of coagulation reactions that culminate in the formation of a fibrin clot. Although the basic process is similar, there are differences in platelet structure, plasma levels of coagulation factors, and physiological platelet response among mammalian species.

Elephant blood clots rapidly but clot retraction is variable. (Clotted whole blood should retract from the sides of a glass tube within 1–2 hours—a crude estimate of platelet function).[34,47,85] Whole blood coagulation time is 3–7 minutes.[66,85]

Elephant platelets have large cytoplasmic granules and lack the well-developed canicular system typical of other mammals. Elephant platelets most resemble those of the bovine in structure and function.[34] Both elephant and bovine platelets are unresponsive to platelet inhibition by acetylsalicylic acid (aspirin).[33] Asian elephant platelets aggregate reversibly, similar to those of killer whales and cattle and in contrast to those of humans and canids.[23]

Asian elephant platelets typically range in number from 80–400 $\times 10^3$/µl,[76] although higher values (637 $\times 10^3$/µl) have been reported.[47] A range of 229–622 $\times 10^3$/µl has been reported for African elephants.[30] One study reported counts as low as 46 $\times 10^3$/µl.[17] In most species, platelet counts <25 $\times 10^3$/µl may result in hemorrhage. If the platelet count is >50 $\times 10^3$/µl and hemorrhages are observed, disseminated intravascular coagulation should be considered.[29]

**Table 25.2.** Coagulation Parameters in Elephants and Horses

| Test | Horses[a] | Elephants |
|---|---|---|
| Platelet count ($\times 10^3$/µl) | 100–600 | 200–600[b] |
| Prothrombin time (sec) | 9.5–13.5 | 9.6 ± 0.7[c] (8.2–10.4) |
| Activated partial thromboplastin time (sec) | 39–64 | 65.6 ± 9.2[c] (52.1–83.5) |
| Fibrinogen (mg/dl) | 200–400 | 461 ± 49[c] (401–546) |

References:
[a]Morris 1999.
[b]Lewis 1974, du Plessis 2002.
[c]Gentry 1996.

In the extrinsic pathway, the conversion of factor VII to its activated form (factor VIIa) initiates the secondary hemostasis cascade. Using factor V as a cofactor, factor VIIa converts factor X to Xa and factor Xa converts prothrombin to thrombin. Prothrombin time, a measure of the integrity of the extrinsic pathway, is 9.6–10.3 seconds in Asian elephants, comparable to human values.[34]

The intrinsic system is initiated with the activation of factor XI by thrombin. Factor XI activates factor IX to IXa; factor IXa activates X to Xa using factor VIIIa as a cofactor. The activated partial thromboplastin test (APTT), a measure of the intrinsic system averages 65.6 ± 9.2 sec.[34] Coagulation studies conducted using commercially available human reference plasma have shown coagulation protein factors VII, IX, X, and XI and antithrombin (AT) in Asian elephants to be similar to human values; however, factor VIII:C is twice as high.[34]

The ability of elephant platelets to secrete granular contents without the formation of irreversible aggregates has been suggested to account for the low reported incidence of thromboembolic disorders in the elephant.[23] Coagulation parameters are summarized in Table 25.2.

## Respiratory Characteristics of Blood

Respiratory characteristics are defined by a number of parameters, including the oxygen capacity, the oxygen dissociation curve, the carbon dioxide dissociation curve, the buffering capacity, the Bohr effect (the effect of pH changes on the oxygen dissociation curve), and the Haldane effect (the effect of oxygen saturation changes on the carbon dioxide dissociation curve).[7] These in turn relate to erythrocyte morphology, body size, and electrolytes within erythrocytes and plasma.

Respiratory function studies conducted with a variety of species have shown a negative correlation between body size and oxygen consumption (the smaller

the animal, the higher the rate of oxygen consumption) and a positive correlation between body size and oxygen affinity (the larger the animal, the greater the affinity of its blood for oxygen).[27] Elephant blood has the highest affinity for oxygen among the species thus far studied.[7,27] The oxygen dissociation curve is similar for both species; however, $P_{50}$ (the partial pressure of oxygen at which 50% of hemoglobin is saturated with oxygen) is higher in Asian elephants.[27]

Compared to other mammals, elephant Hb has a high affinity for oxygen (low $P_{50}$) and a small Bohr effect.[7,27] The blood of both species possesses a single major Hb component, but that of Asian blood migrates slower electrophoretically.[27,82]

Although it has been suggested that lateral recumbency in elephants may result in clinically significant arterial hypoxemia,[38] further studies show that arterial $O_2$ content does not change significantly despite decreases in arterial $O_2$ pressure ($Pa_{O2}$) in elephants maintained in lateral recumbency for 15 minutes.[40] In contrast to most mammalian species, elephants are more safely maintained in lateral recumbency during anesthesia, and sternal recumbency can be fatal. See further discussion in Chapter 9.

The amino acid substitutions in elephant hemoglobin that enhance affinity for oxygen also enhance affinity for carbon monoxide (CO).[8,42] The detrimental effects of CO on mammalian fetuses have been well documented. Exposure to high CO levels (from prolonged housing indoors) combined with an increased affinity of elephant blood for CO has been suggested as a possible explanation for the stillbirths and neonatal deaths experienced by some North American zoos.[6] Further research is needed.

## White Blood Cell (WBC) Morphology and Indices

White blood cell counts vary between 5 to 26 $\times$ $10^3$/µl in most reports, with similar values reported for African and Asian elephants. WBC counts may be higher in young African and Asian elephants.[59,97] There do not appear to be seasonal or ecological variations[17] or gender differences[59,75]; however, one study noted higher WBC counts in free-ranging African females compared to males.[26] No differences have been noted between captive and free-ranging Asian elephants.[76] From a clinical standpoint (in the author's opinion) WBC counts above 18–20 $\times$ $10^3$/µl should be considered elevated.

The lymphocyte is most frequently cited as the predominant cell. Confusion in cell nomenclature has arisen, however, because of an unusual bilobed cell. This cell has been variously classified as a lymphocyte,[17,59,79] a monocyte,[26] or a bilobed monocyte,[3] or its classification has not been clearly stated, making it difficult to compare differential counts between studies. Silva[75] proposes that there are two types of monocytes—a typical unsegmented monocyte and one with a bi- or trilobed nucleus. The presence of peroxidase-positive cytoplasmic granules differentiates these monocytes from lymphocytes. Neutrophils are more accurately described as heterophils because of the presence of reddish cytoplasmic granules with Leishman staining.[75] Monocyte cytoplasmic granules are not apparent with Leishman staining and can thus be differentiated from heterophils.[75] Segmented monocytes occur four times more frequently than unsegmented monocytes. White blood cells are illustrated in Figures 25.3 to 25.14 (Color Section).

Basophils occur but in low numbers.[17,26] Eosinophilia, associated with parasite infections in other species, does not appear to be common even in free-ranging elephants that would be expected to have high parasite loads[26] and in elephants positive for microfilaria.[17] Decreases in lymphocytes and eosinophils have been noted in pregnant Asian elephants.[59] The percentage of B and T lymphocytes seems to be higher in elephants than in horses, based on one study.[89]

It is unknown whether excitement-induced physiologic neutrophilia (pseudoneutrophilia) occurs in elephants as it does in young, healthy horses; however, this has been suggested to explain the somewhat higher WBC counts observed in immobilized free-ranging Asian elephants.[76] Also questionable is whether elephants experience corticosteroid-induced neutrophilia. Typically associated with conditions that cause endogenous corticosteroid release (e.g., pain) or with exogenous corticosteroid administration, corticosteroid-induced neutrophilia is characterized in other species by neutrophilia, lymphopenia, and eosinopenia.[29] In one study, no changes in CBC (or serum chemistry) values were seen pre- and postsemen collection in a manually restrained bull elephant, although serum hydrocortisone levels varied.[88]

Elevations in mammalian WBC counts may be associated with inflammation, infections (bacterial, viral, fungal, or parasitic), tissue necrosis, hypersensitivity, endotoxemia, poisoning, and malignancy. Low WBC counts may be seen with viral diseases; endotoxemia; acute, purulent bacterial inflammation; and certain blood parasites. See Table 25.3 for suggested WBC reference values. The clinicopathologic correlations of spe-

**Table 25.3.** White Blood Cell (WBC) Reference Values

| Parameter | Absolute Range ($\times$ $10^3$/µl) | Relative Range (%) |
|---|---|---|
| WBC | 10–18 | — |
| Heterophils | 2–4 | 25–30 |
| Lymphocytes | 5–8 | 30–40 |
| Monocytes | 2–4 | 25–30 |
| Eosinophils | 0.1–1 | <5 |
| Basophils | 0.00–.03 | <1 |

References: Silva 1993a, b; Nirmalin 1967; Jainudeen 1971; Ratnasooriya 1990; Allen 1985.

cific elephant diseases are discussed further in Chapter 11 and in the appropriate body systems chapters.

## DISEASES AND DISORDERS

### Anemia

Anemia is an absolute decrease in PCV, Hb, and/or RBC count. The RBC count, PCV, and Hb should be interpreted concurrently to assess the status of anemia. Anemia is not a specific diagnosis, and the underlying etiology should be determined and corrected if possible. Associated clinical signs, related to hypoxia and/or hemolysis, may include pale mucous membranes, weakness, dyspnea, exercise intolerance, tachycardia, heart murmur, hypersensitivity to cold, icterus, hemoglobinuria, hemorrhage, and fever. Anemia is not always clinically obvious, particularly in chronic cases where physiological adaptation to reduced oxygen carrying capacity has occurred. The PCV will confirm whether anemia is present. A value of less than 33% has been suggested to define anemia in the elephant;[97] however, a range of 25–44% is considered normal for Asian elephants in Sri Lanka (personal communication, Dr. I. D. Silva, Sri Lanka, July 2005).

Anemia may be classified according to RBC size and Hb concentration, bone marrow response, or pathophysiology. Reticulocytes have not been reported in the literature but have been observed in anemic Asian elephants (personal communication, Dr. I. D. Silva, Sri Lanka, July 2005). Causes of anemia of potential concern for elephants are listed in Table 25.4.

Anemia has been reported in association with liver fluke infection,[20] retained placenta,[57] tuberculosis,[70] tuberculosis treatment,[53] and malabsorption syndrome.[25]

Iron deficiency anemia in three newly imported Asian elephants was treated successfully with iron dextran injections and oral iron sulfate syrup.[45,46]

**Blood groups and blood transfusions.** The author is unaware of any reports of blood groups or successful transfusions in elephants. Before a decision is made to administer a transfusion, the cause, severity, and progression of the anemia must be considered as well as the risk of an adverse reaction. The blood volume of most domestic species is between 6–11% of body weight or 50–110 ml/kg.[2] A figure of 72 ml/kg is commonly used for the horse.[69] A blood volume of 112.6 l measured in a 3216 kg Asian elephant represented only 3.5% of the body weight (35 ml/kg),[73] possibly due to debilitation at the time of death or technical difficulties in sample collection. A sudden loss of one-third of the blood volume will result in shock in most mammals.[29]

Mild-to-moderate chronic anemia may benefit more from identification and correction of the underlying etiology than from a blood transfusion. Even acute blood loss often responds more favorably to restoration of blood volume with intravenous fluids than to replacement of RBCs.

**Table 25.4.** Possible Causes of Anemia in Elephants[a,b]

| Blood Loss | Blood Destruction | Decreased or Ineffective Production |
|---|---|---|
| **Acute** | infections (e.g., clostridium) | chronic renal disease |
| trauma/surgery | RBC parasites (babesia, trypanosoma) | other chronic disease or inflammation (e.g., TB) |
| anticoagulant rodenticide toxicity | hepatic failure | lead poisoning |
| GI ulceration/ hemorrhage | snake bite | metabolic or endocrine disease |
| disseminated intravascular coagulation (DIC) | DIC | neoplasia |
| | toxicities | cytotoxic bone marrow damage (e.g., phenylbutazone) |
| **Chronic** | | |
| parasitism (strongylosis, liver flukes) | | |
| GI ulceration/ hemorrhage | | |
| hematuria | | |
| vitamin K deficiency | | |
| neoplasia | | |
| thrombocytopenia | | |

[a,b]References: Duncan 1994, Aird 2000.

In cases where anemia is severe (Hct <12% and decreasing) and where a transfusion may be life saving, major and minor cross matches should be performed. Mix donor RBCs with recipient plasma for a major cross match and mix recipient RBCs with donor plasma for a minor cross match. Agglutination or lysis in either case indicates an unacceptable match. Consult large animal veterinary or hematology texts for details of cross-matching procedures.[95]

### Polycythemia

Polycythemia (an increase in PCV, RBC count, and Hb) may be primary (associated with myeloproliferative disorders) or secondary (associated with high altitude, chronic pulmonary disease, or cardiovascular anomalies).[29] In the horse, splenic contraction caused by excitement-induced epinephrine release causes massive numbers of RBCs to be injected into the peripheral blood, elevating the PCV as much as 40% and resulting in a relative polycythemia.[44] The increased RBC numbers enhance the oxygen-carrying capacity of the blood but also increase blood viscosity and cardiac load. It is not known whether this phenomenon occurs in elephants. A relative polycythemia may occur with dehydration in any species. Polycythemia (PCV >66%) has been observed in samples collected from culled African elephants[97] and may have been due to acute traumatic shock or to underlying cardiovascular disease.

### Blood Parasites

#### Trypanosomiasis (Surra, Thut).

Definition. Trypanosomiasis is a protozoan disease prevalent in tropical-climate countries. It occurs in a

wide range of hosts, including elephants, horses, cattle, sheep, goats, buffalo, donkeys, mules, monkeys, dogs, cats, pigs, and camels. The disease in humans is known as *sleeping sickness* in Africa and is transmitted by the tsetse fly. In South and Central America, Chagas disease is the human form, and it is transmitted by reduviid species (assassin bugs).

Etiology. *Tryanosoma evansi* in Asian elephants; *T. congolense*[51] and *T. brucei* in African elephants.[18,37]

Epizootiology. The parasite is transmitted by biting flies, including Tabanids, Stomoxys, and mosquitoes. It is most prevalent in Asia during the rainy season.

Clinical signs. In elephants, fever is the earliest sign, but it may go undetected. Lethargy, weakness, exercise intolerance, dull eyes, lacrimation, and dry skin may be noted. The appetite is usually normal. There is a progressive loss of body condition accompanied by profound weakness, anemia, intermittent fever, and debility. Stools may be normal, or constipation alternating with diarrhea may occur.[51,90]

Diagnosis. Organisms are usually plentiful during episodes of fever and can be seen in fresh or stained blood smears. During intermission or in elephants with subclinical infections, when parasites may be few in number, temperature monitoring and daily blood smears may aid detection.[51] An ELISA test has been used to detect infection in African elephants.[43] Elephants that die of surra are typically emaciated. Internal organs are pale and petecchial and ecchymotic hemorrhages may be seen. The abdomen may contain a large amount of straw-colored fluid and the heart may be pale and flabby.[51]

Differential diagnosis. Other parasitic infections, tuberculosis, chronic diseases or conditions causing hemolytic anemia.

**Management.** Separate infected elephants and control fly vectors. Avoid contact with domestic animals that may be infected or carriers. Therapeutic agents reported to be effective in elephants are 1) diminazene aceturate, 3.5 mg/kg deep IM; 2) quinapyramine sulphate, 5 mg/kg SQ; 3) quinapyramine prosalt, 7.4 mg/kg; and 4) antricide methyl sulphate, 2–3 mg/kg.[90] Diminazene acetuarate given at 5–8 mg/kg SQ is also reported to be effective.[22]

**Babesiosis (Piroplasmosis, tick fever).**

Definition. Babesiosis is a protozoan disease that affects humans and domestic and wild animals. It has been reported rarely in both Asian and African elephants.[12,51] Frequent bathing of captive elephants may account for the low prevalence of this and other tick-transmitted diseases even in endemic areas (personal communication, Dr. Krishnamurthy, India, 2000). Parasites similar to *Babesia gibsoni* have been observed in the erythrocytes of three elephants in Sri Lanka. All three responded well, clinically, to treatment with diminazine aceturate (3.5–7mg/kg) together with hematinics (personal communication, Dr. I. D. Silva, Sri Lanka, July 2005).

Etiology. *Babesia spp.*

Epizootiology. Babesiosis has a wide distribution, particularly in the tropics. It is transmitted by ticks (genera and species vary with geographical location).

Clinical signs. Weakness, fever, jaundice, constipation, hemoglobinuria.[51]

Diagnosis. Characteristic organisms on blood smear.

Differential diagnosis. Same as for surra.

Management. Tick removal or dipping. Berenil (Hoechst) (diminazene acetuarate), 5–8 mg/kg SQ (personal communication, Dr. Apurba Chakraborty, India, March 2005).

**Filariasis.**

Definition. *Filaria* is an old generic term for a group of nematode parasites that live in the tissues of vertebrate hosts. Filariasis denotes the presence of microfilaria in the blood and tissues. Elephantiasis, a filarial disease of man, is so named because of the elephantlike appearance of the limbs resulting from circulatory and lymphatic obstruction. Filarial parasites of elephants are described below.

Etiology. In African elephants: *Dipetalonema loxodontis* and *D. gossi*.[5] In Asian elephants: *Indofilaria parabiramani* and *Dipetalonema asiatica*,[4,71] also *Stephanofilaria* sp.[1,10,92]

Epizootiology. Blood-sucking parasites are presumed to be the intermediate hosts. See Chapter 12.

Clinical signs. Clinical signs in African elephants have not been described because samples have been collected opportunistically during culls. However, severe thrombophlebitis and parenchymal liver lesions suggest that *Dipetalonema* sp. cause hepatic disease.[9] In Asian elephants, *Indofilaria pattabhiramani* and *Indofilaria elephantis* are responsible for a cutaneous filariasis that causes 1–2 cm nodules on the sides, lower abdomen, and limbs.[22] *Stephanofilaria* sp. may cause lesions on the back and ventral surfaces ranging from 2.5×17.5 cm. in diameter.[10] Ulcerated, pruritic toe and heel lesions have also been observed.[91,92] Microfilaria are commonly found in free-ranging and domesticated elephants in Sri

Lanka, and those elephants in poor body condition may also demonstrate loss of appetite, lethargy, submandibular anemia or corneal opacity (personal communication, Dr. I. D. Silva, Sri Lanka, July 2005).

Diagnosis. Microfilaria in African elephants may be detected by examination of fresh blood or thick, Giemsa-stained smears. In one study 73.5% of 195 African elephants were infected with *M. loxodontis* (presumably the filarial form of *Dipetalonema* although the location of adult parasites was not investigated). The highest infection rate occurred in the 5–15-year-old age group and the mean number of microfilaria was 289 ± 589/ml of blood.[98] In the case of cutaneous filariasis in Asian elephants, microfilaria can be detected in the blood that oozes from ruptured nodules.[22] Microfilaria may also be seen in peripheral blood; higher counts are detected in nocturnal samples (9 p.m. to 3 a.m.) compared to those collected during the day (personal communication, Dr. I. D. Silva, Sri Lanka, July 2005). See further discussion in the Myanmar section of Chapter 35.

Differential diagnosis. Other parasitic infections, fly bites, abscesses.

Management. Anthiemalin at a dose of 50 ml/2000 kg administered subcutaneously in the neck or tail fold has been found to be effective against cutaneous filariasis in Asian elephants.[22] Stephanofilarial dermatitis has been successfully treated with the topical application of 8% metrifonate (trichlorfon) ointment combined with Himax® (Indian herbal extract cream) for 15 days.[91,92]

**Blood parasites of minor importance.**

Schistosomiasis (Bilharziasis). Schistosomiasis is caused by a trematode; adult flukes reside in the veins. Man is the principle reservoir, although there are numerous animal hosts. It occurs in Africa, South America, the Middle East, and parts of Asia. Infection is acquired from water containing larval forms that have developed in snails. Schistosomiasis was tentatively diagnosed in six elephants showing inappetance, constipation, diarrhea, and vomition. Large numbers of eggs were observed in the feces but they differed in size from a previous report and a definitive identification was not established.[65]

Other. Certain blood parasites have not been reported in elephants but may be of potential concern where there is close proximity to domestic livestock. These include Anaplasma, an intraerythrocytic ricketssia of cattle, sheep, and goats, and Theileria, a protozoal parasite that infects erythrocytes and lymphocytes of ruminants.

### Lymphatic Disorders

Lymphoid nodules develop in the lungs of African elephants associated with herpesvirus,[50] and similar lesions have been noted in the pancreas.[9] Eosinophilic and granulomatous lymphadenitis are associated with hepatic dipetalonemiasis in African elephants.[9]

## SERUM CHEMISTRIES

### Introduction

Serum chemistries are an important diagnostic tool for elephants, especially when clinical signs are nonspecific and other diagnostic techniques may not be available. Serum chemistry values may be altered by environmental and physiological factors, restraint techniques, specimen handling, laboratory methodologies and quality control, and numerous other variables.

Studies of captive elephants may select for variables such as age or gender and samples are generally collected without sedation, whereas studies of free-ranging elephants are usually opportunistic and samples collected during immobilization or culling. Most chemistry panels are designed for humans or domestic animals, and not all tests are applicable to elephants. Nonetheless, sufficient studies have been conducted to establish "reference" if not "normal" values for elephants and to engender confidence in using these to monitor the clinical course of disease, especially if sequential samples are obtained and submitted to the same (reliable) laboratory.

Many reports cite variations that although statistically significant may not be clinically relevant. Biochemical test values that fall outside the reference range are useful only insofar as they have a high degree of correlation with the presence of specific organ malfunction. Although such correlations have not been extensively evaluated for elephants, information from domestic species provides a useful guideline to use in conjunction with history, presenting signs, physical examination, and other diagnostic tests. Table 25.5 describes serum chemistry tests, summarizes causes of elevated and decreased values, and suggests reference ranges for elephants.

Chemistry values of healthy individual elephants are generally consistent over time although random changes in cholesterol may occur.[58] Elevations in alkaline phosphatase, gamma glutamyl transferase, and creatinine have been associated with musth.[58] Values are similar between captive and free-ranging Asian elephants.[75,76] Age or physiological status (e.g., lactation) may affect certain parameters, but gender differences are few. Seasonal variations in specific chemistries observed in free-ranging African elephants reflect the impact of reduced quantity and quality of food and limited water supply. Dry season values are lower for blood urea nitrogen (BUN), potassium, phosphorus, albumin, and thyroxin and higher for creatinine, sodium, globulins, and $T_3$ uptake.[14,15,16]

Baseline samples obtained on individual captive elephants while healthy may provide the most accurate reference values when illness is suspected. Com-

**Table 25.5.** Chemistry Reference Values

| | Units[d] | Reference Range Horses[a] | Reference Range Elephants[b] | What It Is | Possible Causes of High Values | Possible Causes of Low Values | Comments |
|---|---|---|---|---|---|---|---|
| Alanine aminotransferase (ALT); previously Serum glutamic pyruvic transaminase (SGPT) | IU/L | | 1.5–3.0 | Enzyme that catalyzes the transamination of L-alanine and 2-oxoglutarate to pyruvate and glutamate; muscle specific in large animals; liver specific in dogs | May be associated with muscle damage in lambs, pigs, and horses; | Levels are normally low in large animals | Not clear whether this is an accurate indicator of muscle damage for elephants |
| Albumin | g/dl | 2.5–4.0 | 1.5–3.5 | Plasma protein made in the liver; maintains plasma colloidal osmotic pressure; transport of vitamins, hormones, hemoglobin, etc. | Uncommon; spurious elevation with dehydration | ↓ Production (malnutrition, malabsorption, chronic liver disease, exocrine pancreatic disease); ↑ loss (renal disease, hemorrhage, burns, parasites) | Lower in elephants than other mammals; higher in wild during the wet season (Brown 1978) |
| Albumin/globulin ration (A/G) | | 0.6–1.5 | 0.2–1.2 | Ratio used to interpret protein alterations | Hypoglobulinemia | Hyperglobulinemia | Elephant ratio lower than most mammals (Nirmalin 1971); significantly lower in TB + elephants (Harr 2001) |
| Alkaline phosphatase (ALP) | IU/L | 100–260 | 60–450 | Multiple isoenzymes; nonspecific; hydrolyze phosphate esters to yield inorganic phosphate; in every tissue; high levels in bone, kidney, liver, intestine, placenta | Cholestasis; bone disease (rickets, neoplasms, fractures, periosteal inflammation); indicator of colostrum absorption | Not significant | Higher levels in neonates; may ↑ in musth (Niemuller 1990); phenylbutazone, phenobarbitol, ketoconazole are known to elevate levels in other species |
| Amylase | | 3–30 IU/L | 380–2755 Somogyi units/100 ml; 148–242 units/ml | Pancreatic enzyme (hydrolyzes 1,4-glycoside linkages to form mono- and disaccharides | Nonspecific; may increase with pancreatic, gi, renal or liver disease | Not significant | Wide range in elephants; probably not a useful test |
| Anion gap | mEq/L | 7–16 | | A calculated value used to evaluate acid-base disturbances; ($[Na^+ + K^+] - [Cl^- + HCO_3^-]$) | Renal disease, diabetes, lactic acidosis, some toxicities | Uncommon; may be seen with low albumin or hemodilution | |

(*continued*)

**Table 25.5.** Chemistry Reference Values (*continued*)

| | Units[d] | Reference Range Horses[a] | Reference Range Elephants[b] | What It Is | Possible Causes of High Values | Possible Causes of Low Values | Comments |
|---|---|---|---|---|---|---|---|
| Aspartate aminotransferase (AST); previously serum glutamic oxaloacetic acid (SGOT) | IU/L | 150–300 | 15–35 | Enzyme that catalyzes the transamination of L-aspartate and 2-oxoglutarate to oxaloacetate and glutamate; found in almost all tissues, but highest levels in liver and cardiac and skeletal muscle | Degenerative or necrotizing muscle damage due to inflammatory, traumatic, or degenerative conditions such as Clostridial myositis, systemic infections, muscle trauma, transport or capture myopathy, prolonged recumbency, or bacterial endocarditis | Not significant | CK and LDH will also ↑ with muscle damage; the half-life of AST is longer than CK in other species (12 hrs in dogs, 18 hrs in swine, and probably longer in horses) so ↑ AST in the absence of ↑ CK or clinical signs related to muscle damage, suggests liver damage |
| Bile acids | µmol/L | <15 | 6–15 | Steroid acids synthesized in the liver from cholesterol; aid in fat digestion and absorption in the intestine | Liver disease, cholestasis | Not significant | No postprandial increase in horses nor likely in elephants due to lack of a gall bladder |
| Bilirubin, total | mg/dl | 0.5–2.0 | 0.2–1.0 | Hemoglobin breakdown product; present in conjugated and unconjugated form in serum | Liver disease, hemolytic anemia | Not significant | |
| Bilirubin, direct (conjugated) | mg/dl | 0.0–0.4 | .03–.80 | Bilirubin that is conjugated in the liver (with glucuronic acid primarily) and secreted into bile | Liver disease; cholelithiasis; cholangiohepatitis (e.g., liver flukes) | Not significant | |
| Bilirubin, indirect (unconjugated) | mg/dl | 0.5–1.7 | 0.0–0.6 | Albumin-bound prehepatic bilirubin | Liver disease, hemolytic anemia | Not significant | |
| Blood gases—Arterial (upright) | | | | | | | |
| pH | | 7.38–7.44 | 7.40 ±0.01 [c] | Hydrogen ion concentration; a measure of acidity or alkalinity | **Metabolic alkalosis:** $Cl^-$ or $K^+$ depletion; ileus; diuretics **Respiratory alkalosis:** pulmonary disease; heart failure; severe anemia | **Metabolic acidosis:** acute diarrhea; hypovolemic shock; gi torsion; peritonitis **Respiratory acidosis:** pulmonary disease; inadequate ventilation during general anesthesia; opiates | Consult appropriate large animal or clinical pathology texts for additional information |
| $HCO_3^-$ | mEq/l | 23.6–27.0 | 23.6 ±0.6[c] | Anion in plasma | Metabolic alkalosis; compensatory response to respiratory acidosis | Metabolic acidosis; compensatory response to respiratory alkalosis | |

**Table 25.5.** Chemistry Reference Values (*continued*)

| | Units[d] | Reference Range Horses[a] | Reference Range Elephants[b] | What It Is | Possible Causes of High Values | Possible Causes of Low Values | Comments |
|---|---|---|---|---|---|---|---|
| $PCO_2$ | mmHg | 38.2–43.4 | 39.4 ±0.3[c] | Partial pressure of carbon dioxide | Respiratory acidosis; compensatory response to metabolic alkalosis | Respiratory alkalosis; compensatory response to metabolic acidosis | |
| $PO_2$ | mmHg | 95–100 | 103 ±2[c] | Partial pressure of oxygen | | | |
| Blood gases— Venous (upright) | | | | | | | |
| pH | | 7.36–7.40 | 7.36 ± 0.01[c] | | | | |
| $HCO_3^-$ | mEq/l | 23.7–27.1 | | | | | |
| $PCO_2$ | mmHg | 40.4– 45.6 | 46.8 ± 1.9[c] | | | | |
| Total$CO_2$ | mEq/l | 26–35 | 20–28 | Comprised of ~95% $HCO_3^-$; a crude measure of acid-base status | Metabolic alkalosis | Metabolic acidosis | Blood gas determination preferred to evaluate acid-base disorders; significantly higher in TB + elephants (Harr 2001) |
| Blood urea nitrogen (BUN) | mg/dl | 13–26 | 5–20 | Main nitrogenous end product of protein metabolism formed in the liver from ammonia and amino acids; excreted mainly by the kidney; intestinal excretion in the horse | Renal disease; shock, dehydration, cardiovascular disease | Hepatic insufficiency; low protein diet; anabolic steroids; may be normal in young animals (true for elephants) | Mild ↑ may be seen with starvation, heavy exercise, fever, infections, steroids |
| Calcium | mg/dl | 10–13 | 9–12 | Cation essential to intra- and extracellular fluid, blood clotting, and various neuromuscular and metabolic activities; comprised of ionized (~50%), protein bound (~40%) and complexed (~10%) calcium | Hyperalbuminemia; chronic renal failure; certain plant intoxications; neoplasia | Hypoproteinemia (hypoalbuminemia); acute renal failure; acute toxemia or septicemia; transport tetany; capture myopathy | Significantly higher in TB + elephants (Harr2001); may be higher in baby elephants (Sreekumar 1989b); levels <6 mg/dl will cause recumbency in most species; <4 mg/dl may be fatal |
| Calcium, ionized | mg/dl | | 3.9–5.3 | Physiologically active form of calcium | Alkalosis | Acidosis | Ionized calcium will generally remain normal when total Ca is altered by changes in protein levels |
| Chloride | mEq/l | 98–109 | 100–115 | Electrolyte (anion) essential to the normal function of all cells | Water deprivation; salt poisoning; diarrhea, diuretics, diabetes, burns | Diarrhea; ascites; peritonitis; hemorrhage; spurious ↓ with hyperlipidemia, hyperproteinemia, hyperglycemia | Changes usually parallel changes in Na resulting from altered water balance; varies inversely with $HCO_3$ |

(*continued*)

**Table 25.5.** Chemistry Reference Values (*continued*)

| | Units[d] | Reference Range Horses[a] | Reference Range Elephants[b] | What It Is | Possible Causes of High Values | Possible Causes of Low Values | Comments |
|---|---|---|---|---|---|---|---|
| Cholesterol (total) | mg/dl | 50–150 | 26–68 | A steroid alcohol found in animal fats; essential component of cell membranes, bile acid, and steroid hormone precursor | May increase secondary to liver, renal, or endocrine disease | Not significant | Inverse relationship to thyroid activity; arteriosclerotic disease in free-ranging African elephants has not been associated with elevated cholesterol, phospholipids, triglycerides, or free fatty acids (Dillman 1970; McCullagh 1972) |
| Cortisol, baseline | µg/dl | 3.0–6.0 | | Hormone secreted by the adrenal cortex | | | Use ACTH stimulation or dexamethasone supression tests to diagnose adrenal dysfunction |
| Creatinine kinase (CK) | IU/L | 60–330 | 50–250 | An enzyme that catalyzes the transfer of a high-energy phosphate bond from ATP to creatine in muscle; one of the most organ-specific enzymes; highest levels in skeletal and cardiac muscle and brain; lesser amounts in intestines, uterus, urinary bladder, and thyroid | Same as AST | Not significant | Also called creatine phosphokinase (CPK); hemolysis may cause false ↑; injections may cause ↑ up to 1 week postinjection; CK has a short half-life in serum; values return to normal with 2–3 days after muscle necrosis ceases; persistent high levels indicate active muscle disease (see AST) |
| Creatinine | mg/dl | 1.0–2.0 | 1.0–2.0 | End product of creatine metabolism in muscle; moves from muscle to blood and is excreted by the kidney | Renal disease | Not significant | May ↑ with musth (Niemuller1990); may be lower in young elephants (Senthilkumar 1999) |
| Fibrinogen | mg/dl | 100–400 | 100–400 | Plasma protein formed in the liver; aids in blood clotting (converted to fibrin by thrombin) | Nonspecific indicator of inflammation or neoplasia; spurious increase with dehydration | Liver disease; prolonged thrombin time; sequel to disseminated intravascular coagulation (DIC); (may be normal or elevated if DIC results from inflammatory disease) | Not very well studied in elephants; values >1000 mg/dl signify a poor prognosis in horses; differentiate true ↑ from dehydration by plasma protein/fibrinogen ratio: PP/F >15 = dehydration; PP/F <10 = elevated fibrinogen |

**Table 25.5.** Chemistry Reference Values (*continued*)

| | Units[d] | Reference Range Horses[a] | Reference Range Elephants[b] | What It Is | Possible Causes of High Values | Possible Causes of Low Values | Comments |
|---|---|---|---|---|---|---|---|
| Gamma glutamyl transferase (GGT) | U/L | 9–25 | 4–35 | Enzyme involved in glutathione metabolism; found in most cells; highest activity in liver and kidney | Cholestasis; acute hepatic necrosis; indicator of colostrum absorption | Not significant | Not very well studied in elephants; may ↑ with musth (Niemuller 1990) |
| Globulin, total | g/dl | 2.6–4.0 | 3.7–6.5 | Plasma proteins that function as carriers, clotting factors, complement components, acute phase reactants, and immunoglobulins; can be separated into 5 fractions | Inflammation; necrosis; surgery; tumors; chronic infections; abdominal or pulmonary abscess | Failure of passive transfer, immunodeficiencies, hemorrhage, exudation; may ↓ with albumin in cases of malnutrition, malabsorption, hemorrhage | Globulins higher in elephants than other mammals; lower in the wet season in wild African elephants and may vary geographically (Brown 1978) |
| α1 | g/dl | 0.1–0.7 | 0.7–0.8 | Synthesized by the liver; acute phase reactant | Tissue injury or inflammation | | α Globulins may be lower in elephants 0–2 yrs (Brown 1978) |
| α2 | g/dl | 0.1–0.7 | 0.7–0.8 | Synthesized by the liver; acute phase reactant | Tissue injury or inflammation | | |
| ß1 | g/dl | 0.4–1.6 | 0.4–0.5 | Synthesized by the liver | Generally associated with high γ globulins | | |
| ß2 | g/dl | 0.3–0.9 | 0.6–0.8 | Synthesized by the liver | Generally associated with high γ globulins | | |
| γ | g/dl | 0.6–1.9 | 2.9–3.4 | Secreted by B lymphocytes and plasma cells | Chronic antigenic stimulation (e.g., chronic hepatitis or liver abscess); neoplasia; immune-mediated diseases | | May be lower in elephants 0–5 yr (Brown 1978) |
| Glucose | mg/dl | 76–127 | 60–116 | End product of carbohydrate digestion; provides energy for body cells; excess stored as glycogen; regulated by insulin and glucagon | Acute colic; stress; administration of glucocorticoids or xylazine | Septicemia, endotoxic shock; liver failure; inappetance in newborn animals | Significantly lower in TB + elephants (Harr 2001) |
| Iron (serum, SI) | µg/dl | 73–140 | 60–150 | Essential component of hemoglobin; transported in the blood by transferrin, a ß-globulin | Hemolytic anemia; glucocorticoid excess (in the horse) | Chronic hemorrhage; acute or chronic inflammation; anemia of chronic disorders (infectious, inflammatory, or neoplastic); iron deficiency | |
| Iron-binding capacity total (TIBC) | µg/dl | 200–262 | | A measure of total serum transferrin | | Iron deficiency | TIBC minus serum iron = the unbound iron-binding capacity |

(*continued*)

**Table 25.5.** Chemistry Reference Values (*continued*)

| | Units[d] | Reference Range Horses[a] | Reference Range Elephants[b] | What It Is | Possible Causes of High Values | Possible Causes of Low Values | Comments |
|---|---|---|---|---|---|---|---|
| Lactate dehydrogenase (LDH) | IU/L | 140–460 | 250–500 | An enzyme (catalyzes the reaction of L-lactate to pyruvate); found in all tissues; muscle, liver, and RBCs are the major sources | Same as AST | Not significant | Hemolysis causes false elevations in dogs but not in horses |
| Lipase | IU/L | 23–87 | 10–90 | Enzyme produced in the pancreas and intestinal mucosa that catalyzes breakdown of fats into glycerol and fatty acids and promotes absorption of fat-soluble vitamins | Acute pancreatitis; may also ↑ in renal failure, hepatic disease and neoplasia | Not significant | May be a more sensitive test for pancreatitis than amylase; dexamethasone will increase lipase activity in dogs |
| Magnesium | mg/dl | 1.4–2.3 | 1.4–2.6 | | Renal failure; may occur with oral or rectal overdose of Epsom salts ($MgSO_4$) or magnesium containing laxative | Grass tetany in ruminants | Abnormalities of greater concern for ruminants |
| Osmolality | mOsmol/L | 270–300 | 250–280 | A measure of the number of dissolved particles per unit of water in serum used to evaluate hydration status | Dehydration or fluid volume deficit | Overhydration or edema | |
| Phosphorus | mg/dl | 3.0–5.5 | 4.0–6.0 | An anion that functions in acid-base balance; in the form of adenosine triphosphate (ATP), it is the main form of energy storage and transfer for numerous metabolic processes | Acute renal failure; vitamin D toxicity; acute rhabdomyolysis | Chronic renal failure; starvation or chronic wasting | Higher levels have been observed in baby Asian elephants (Nirmalin 1969) |
| Potassium | mEq/l | 2.5–4.5 | 3.0–6.0 | Electrolyte (cation) Essential to the normal function of all cells; critical role in acid-base and water balance; must be properly balanced with $Na^+$ and $Ca^+$ in plasma for proper cardiac function | Metabolic acidosis; hypovolemia and renal failure; capture myopathy; spurious ↑ with in vitro hemolysis or prolonged storage of unseparated blood | Diarrhea, torsion, or volvulus; peritonitis; severe inappetance; dietary deficiency | |

**Table 25.5.** Chemistry Reference Values (*continued*)

| | Units[d] | Reference Range Horses[a] | Reference Range Elephants[b] | What It Is | Possible Causes of High Values | Possible Causes of Low Values | Comments |
|---|---|---|---|---|---|---|---|
| Sodium | mEq/l | 136–142 | 120–140 | Electrolyte (cation) essential to the normal function of all cells; concentration gradients of $Na^+$ and $K^+$ across cell membranes facilitate transmission of electrochemical impulses in muscle and nerves | Water deprivation; salt poisoning; diarrhea, diuretics, diabetes, burns | Diarrhea; hemorrhage; ascites; peritonitis; volvulus or torsion; spurious ↓ with hyperlipidemia, hyperproteinemia, hyperglycemia | |
| Thyroxin (T4) baseline | µg/dl | | 113.6 ± 27.0 µmol/l[e] | Hormone secreted by the thyroid gland with regulation by the pituitary; involved in metabolism and growth | | Food deprivation; administration of phenylbutazone | Thyroid disease has not been reported in elephants |
| T3 Uptake | % | | 30.1 ± 3.8%[e] | | | | |
| Total protein | g/dl | 5.2–7.9 | 6–12 | Comprised of albumin, globulin, fibrinogen; the plasma proteins maintain osmotic pressure and acid-base balance; individual proteins function as antibodies, enzymes, hormones, coagulation factors, and transport vehicles; can be separated into 5 bands by electrophoresis | Dehydration (relative) hyperfibrinogenemia (inflammation, neoplasia) hyperglobulinemia (inflammation, necrosis, surgery, tumors) | See hypoalbuminemia, hypofibrinogenemia, and hypoglobulinemia | Higher in elephants than most mammals |
| Triglycerides | mg/dl | 6–54 | 15–60 | Lipid storage form | Postprandial; may be secondary to liver, kidney, pancreatic, or endocrine diseases | | See comments in cholesterol above |
| Zinc | µg/dl | | 203–275 | | | | |

[a]References: Duncan 1994, Pearson 2002, Morris 1999.

[b]References for elephant values: Allen 1985; Niemuller 1990; Silva 1993a, b; Caple 1978; Skinner 1976; Brown 1980; Sreekumar 1989b, 1992; Nirmalin 1971; Ratnasooriya 1995; Nirmalin 1969; Brown 1978; Brown 1980; ISIS 2002.

[c]Isaza 2003; values are for standing elephants.

[d]Conversion to Système International (SI). See Appendix 9.

[e]Pichaicharnarong 1983.

parisons between elephants in a collection or monitoring a disease condition in an individual over time may be more informative if a single laboratory is used (provided that it has a good quality control program). New baseline healthy profiles should be established if laboratories change. Drugs may affect certain laboratory values and appropriate references on this topic should be consulted.[64,81]

## Liver Enzymes and Liver Function Tests

A variety of laboratory tests are used to evaluate liver function. Serum enzymes include aspartate aminotransferase (AST, previously serum glutamic oxaloacetic transaminase or SGOT), alkaline phosphatase (ALP), γ-glutamyl transferase (GGT), alanine aminotransferase (ALT, previously serum glutamic pyruvic transaminase or SGPT), and sorbitol dehydrogenase (SDH). Bilirubin, bile acids, the sulfobromophthalein (BSP) excretion test, and ammomnia are additional tests. The diagnostic and prognostic value of these tests varies among domestic species and not all have been adequately studied in elephants.

**Aspartate aminotransferase (AST).** AST (SGOT) occurs in almost all cells. Levels are highest in liver, cardiac, and skeletal muscle. Increased levels are diagnostic for disease in these tissues, especially in large animals. AST has high sensitivity but not specificity for liver disease. It is more reliably an indicator of liver disease in the absence of clinical muscle damage and if creatine kinase (CK) levels are normal.[29] AST may increase with hemolysis. Levels in elephants vary from 4.22–33.0 U/L.[3,13,17,39,60,87] AST is not affected by age or gender.[87]

**Alkaline phosphatase (ALP).** ALP occurs in all tissues with the highest levels in liver, bone, kidney, intestine, and placenta.[29] Levels are typically higher in young growing mammals, including elephants.[14]

Increases due to hepatic disease result from cholestasis and may precede a rise in bilirubin. ALP is not a sensitive indicator of liver disease in the horse. Levels may increase with periostitis, rickets, and primary and secondary hyperparathyroidism and transiently with fractures.[29]

Nonmusth ALP in an Asian bull was 136.2 ± 24.2 U/L compared to 204.4 ± 39.6 U/L during strong musth.[58] Increases in ALP (up to 100-fold) and GGT are indicative of colostrum absorption in domestic species,[29] and this may be true for elephants. Nonsteroidal drugs such as ketoconazole and phenylbutazone can induce ALP.

**γ-glutamyl transferase (GGT).** GGT is a fairly liver-specific indicator of cholestasis commonly used in horses, pigs, and domestic ruminants. GGT may increase in musth bulls.[58] Nonmusth and musth values for GGT were 1.74 ± 1.6 U/L and 7.89 ± 4.59 U/L, respectively, in an Asian bull monitored through two musths. Large amounts of GGT are found in the colostrum of cows, sheep, and dogs,[29] and with ALP it is an indicator of passive transfer.

**Alanine aminotransferase (ALT).** ALT (SGPT) is liver specific for dogs and cats, but hepatic activity is low in horses, domestic ruminants, and pigs.[29] Serum levels are also low in elephants,[3,13,17,58,60,87] and this is not likely a useful test for elephants.

**Sorbitol dehydrogenase (SDH).** SDH is the most sensitive enzyme for liver disease in the horse. It has a short plasma half-life, however, and samples must reach the lab within 4 hours. Values have not been reported for elephants.

**Bilirubin.** Unconjugated bilirubin is comprised largely of hemoglobin released from aged RBCs. It is bound to albumin and transported to the liver where it is conjugated. Conjugated (direct) bilirubin is secreted into bile, transferred to the intestine, converted to urobilinogen by intestinal bacteria, and excreted. Causes of hyperbilirubinemia include hemolysis, acute or chronic hepatocellular disease resulting in reduced functional mass (fibrosis or necrosis), and intra- or extrahepatic cholestasis or bile duct obstruction. Unconjugated bilirubin predominates in horses with hyperbilirubinemia regardless of etiology, whereas in ruminants unconjugated bilirubin is typical.[29] Elevated total bilirubin (4.94 mg/dl) was observed in a 30-year-old Asian cow with colic resulting from overfeeding of produce. Values for indirect and direct bilirubin were 3.7 mg/dl and 1.2 mg/dl, respectively. The condition resolved with a correction of diet and administration of flunixin (personal communication, Dr. Kelly Helmick, Seattle, Washington, July 2005).

**Bile acids.** Bile acids are synthesized in the liver from cholesterol. They are sequestered in the gall bladder in most species and are released into the intestine to assist with fat digestion and absorption. In horses, elevated bile acids (>14–20 μmol/L) are associated with reduced functional hepatic mass, cholestasis, or portosystemic shunts.[68] Lacking a gall bladder, horses do not show a postprandial increase in bile acids. This is likely true for elephants. Elevated bile acids have been observed in an elephant receiving antituberculosis drugs (personal communication, Dr. Ray Ball, Tampa, Florida, June 2005).

**BSP (Sulfbromophthalein) excretion.** In the BSP excretion (or clearance) test, BSP (a dye) is injected intravenously and measured several times postinjection. In normal horses, the half-life is 3.5 minutes.[62] Reduced clearance is seen in cholestatic conditions. BSP half-times reported in four Asian elephants infected with liver flukes were 3.6, 4.3, 5.1, and 6.5 minutes and presumably reflect varying degrees of damage.[20]

**Ammonia.** Ammonia as a test of hepatic insufficiency is not of practical significance for elephants.

## Kidney Enzymes

Values for blood urea nitrogen (BUN) and creatinine are comparable to other domestic species. Allen found BUN to vary with age in African elephants but did not consider this to be clinically important.[3] Creatinine values may be lower (0.82 ± 0.09 mg/dl) in young elephants[72] and higher in adult musth bulls.[58] As in other species, BUN and creatinine levels will increase in renal failure.[56]

## Muscle Enzymes

Creatine kinase (CK), lactate dehydrogenase (LDH), aspartate aminotransferase (AST), and alanine aminotransferase (ALT) are indicators of muscle disease in domestic large animals. Elevated levels may occur with degenerative or necrotizing injuries, clostridial myositis, rhabdomyolysis, capture myopathy, prolonged recumbency, vitamin E/selenium deficiency, bacterial endocarditis, and aortic thrombosis.[29]

**Creatine kinase (CK).** CK is the most sensitive muscle enzyme. Elevations occur within a few hours of injury, returning to normal within 24–48 hours following cessation of further insult. CK is skeletal and cardiac muscle specific. It has a short half-life (2h in the horse), so continued high levels indicate ongoing muscle disease. Levels of CK in healthy Asian elephants vary from 30 to 50 U/L,[87] with somewhat higher levels reported in performing Asian elephants[47] and samples collected from wild African elephants during a cull.[13]

**Lactate dehydrogenase (LDH).** LDH is present in all tissues, but activity is highest in muscle, liver, and RBCs. There are five isoenzymes. A history of muscle injury and/or elevated CK may help determine whether LDH is of muscle or liver origin.

**Aspartate aminotransferase (AST).** AST (SGOT) is nonspecific, but muscle and liver are primary sources.

**Alanine aminotransferase (ALT).** ALT (SGPT) is considered muscle specific in large domestic animals because hepatic levels are quite low. Increased levels occur in myopathies in horses and other domestic species.[29]

AST, LDH, and CK are not affected by age or gender in the elephant.[87] Reports correlating increased muscle enzymes with disease in elephants are lacking. The author, however, witnessed a case of capture myopathy in a wild Asian elephant following capture and a prolonged translocation attempt over a 5-day period. See Table 25.6. Capture myopathy is discussed further in Chapter 17.

CK and LDH display isoenzymes that may have tissue specificity. These should be investigated in cases of known or suspected cardiac or skeletal muscle disorders in elephants.

**Table 25.6.** Serum Chemistries in an Asian Bull with Capture Myopathy

| Test | Reference Value[a] | 3 Days Postcapture | 7 Days Postcapture |
|---|---|---|---|
| AST (U/L) | 15–35 | 503 | 1,197 |
| ALT (U/L) | 1.5–3.0 | 80 | 194 |
| CK (U/L) | 50–250 | 184 | 85,360 |
| BUN (mg/dl) | 5–20 | 16 | 21 |
| Creatinine (mg/dl) | 1.0–2.0 | 3.47 | 5.01 |

[a]See Table 25.5.

## Proteins

Elephant blood is higher in total protein and lower in albumin and has a lower albumin/globulin level than most mammals. There are five main protein bands.[17,35,47,83] The viscosity of elephant blood is almost twice that of other domestic species, a phenomenon attributed to marked agglutination and high levels of globulins.[93]

Albumin values are similar for captive Asian and African elephants, ranging from 1.20–3.62 g/dl.[3,83] Albumin levels may decrease in wild African elephants during the dry season, and higher levels (3.42–4.10 g/dl) have been recorded in African elephants during a cull.[15] Significant decreases in total protein or albumin are unlikely to occur unless reduced protein intake is severe or prolonged.[15]

The A/G ratio is <1 in Asian elephants but may be slightly higher in Africans.[3,83] Globulin values vary from 3.71–6.50 g/dl.[83] Elephants culture positive for tuberculosis have demonstrated lower A:G ratios when compared to previous individual baseline values.[36]

Fibrinogen, an acute phase protein produced by the liver and associated with inflammation in the horse, has been poorly studied in the elephant. Low fibrinogen may indicate liver failure or disseminated intravascular coagulation. Fibrinogen levels >1000 mg/dl denote a poor prognosis in horses. Fibrinogen may be lower in nonlactating versus lactating female elephants.[61]

## Minerals and Electrolytes

In a comparative study, calcium (Ca), magnesium (Mg), and chloride values were similar for baby (<15 years), tuskers, and adult lactating and nonlactating female Asian elephants[60] but a later study showed higher Ca higher in baby elephants.[84] Age-related differences in chloride levels were noted in captive African elephants but were not considered important.[3] Inorganic phosphate is higher in baby Asian elephants.[60] Electrolyte values are similar to other species but may vary seasonally.

Calcium is involved in numerous critical metabolic processes, including muscle contraction, conduction of nerve impulses, cellular membrane permeability, blood coagulation, and others. It is of particular concern for young elephants because an imbalance may result in metabolic bone disease (rickets). It is important to note

that serum calcium is maintained within a fairly narrow range by the body's regulation of intestinal absorption, renal excretion, and mobilization from bone. Serum calcium levels are not a reliable indicator of adequate dietary calcium and will decrease only when the abovementioned regulatory mechanisms are compromised.

## Lipids

Cholesterol, cholesterol esters, triglycerides, and phospholipids comprise the major lipid types. Values have been published for African[16,17,24,28,47,54] and Asian elephants,[60,67,78,86] but correlations with specific disorders have not been reported.

Mean plasma cholesterol values of 30.2 mg/dl and 33.9 mg/dl in 8 free-ranging and 4 domesticated Sri Lankan elephants, respectively,[78] supported earlier findings, which reported a mean cholesterol of 45.23 ± 1.37 mg/dl in 43 domesticated Sri Lankan elephants.[67] Higher values (~110 mg/dl) in another study[60] may have been due to differences in assay procedure or subspecies variation.

Triglyceride values were 19.5 mg/dl for free-ranging and 10.2 mg/dl for domesticated Sri Lankan elephants[78] and 30 mg/dl for free-ranging African elephants.[54] Total lipid in African elephants was 220 mg/dl.[54]

Although arteriosclerotic disease has been observed in free-ranging African elephants, it has not been associated with elevated cholesterol, phospholipids, triglycerides, or free fatty acids.[28,49] See further discussion in Chapter 24.

Lipemia (a milky appearance of serum or plasma due to an excess of lipids in the blood) may influence results of certain biochemical tests. Values that may be falsely lowered by lipemia include total protein, albumin, sodium, and potassium. Values that may be falsely elevated include glucose, calcium, phosphorus, total bilirubin, and hemoglobin. Placing a lipemic serum sample in the refrigerator may encourage separation of the lipid layer.[29]

## Pancreatic Enzymes

The enzyme α–amylase is often considered an indicator of pancreatitis; however, it has poor specificity and high levels may also occur with gastrointestinal, liver, or kidney disease or the administration of narcotics. The wide range of values reported for elephants may reflect differing methodologies. Levels of 1432 ± 469 Somogyi units/100 ml and 192 ± 29 units/ml were reported using the chromogenic substrate and amyloclastic methods, respectively, in African elephants.[80] Reported values for Asian elephants are 381 ± 20.81 Somogyi units/100 ml (n = 12)[60] and 3114 ± 2417 U/L (n = 78).[39] Lipase is a more sensitive test; however, levels may increase in renal failure. Reported lipase levels are 17 ± 18 U/L for Asian elephants (n = 42) and 10 ± 12 (n = 21) for African elephants.[39] The antemortem diagnosis of pancreatic disease in elephants has not been reported.

## Glucose

In vitro glycolysis will reduce blood glucose levels, so serum or plasma should be separated from cells within 30 minutes or less to insure an accurate value. Alternatively, blood may be collected in a sodium fluoride tube.

Relatively low blood glucose values (52–67 mg/dl) have been reported in Asian elephants,[60] and higher values (92 ± 21 mg/dl for Asians and 84 ± 14 for Africans) have been compiled from captive elephants in North America.[39] Lower glucose levels may occur in elephants that are culture positive for tuberculosis.[36]

In adult horses, hypoglycemia may be associated with hepatic failure or bacteremia; hyperglycemia may reflect stress or pain.[68] Diabetes occurs rarely in horses and has not been reported in elephants.

## Thyroid Function Tests

Thyroxine (T4) and triiodothyroxine (T3) are the main thyroid hormones. They are involved in numerous metabolic functions and are discussed further in Chapter 23. Reported T4 and T3 values for healthy Asian elephants (n = 58) were 113.6 ± 27.0 nmol/l and 1.8 ± 0.7 nmol/l, respectively. Values were highest in elephants <10 years old, declining somewhat with age. T3 uptake in this group was 30.1 ± 3.8%.[63] Lower thyroxine levels have been recorded in free-ranging African elephants in the dry season, coincidental with reduced food quality.[15]

## Elephant Panels

Based on tests known to have significance for elephants (or those worthy of further investigation) a basic clinical chemistry screening panel is recommended in Table 25.7. Commercial panels designed for humans or other species may include additional tests and may be more cost effective. This is acceptable as long as the clinician realizes that not all tests have practical application. Additionally, a comprehensive list of baseline health and infectious disease screening tests is provided in Table 25.8 as a reference for veterinarians conducting international field research with elephants.

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**Table 25.7.** Suggested Basic Elephant Chemistry Panel

| | |
|---|---|
| Total protein | CK |
| Albumin | LDH |
| BUN | Na |
| Creatinine | Cl |
| AST (SGOT) | K |
| ALP | Ca |
| GGT | P |
| Bile acids | Fibrinogen |
| Total bilirubin | |

**Table 25.8.** Comprehensive Elephant Screening Protocol for Baseline Health Evaluation and Infectious Diseases Recommended for International Field Research[a]

| |
|---|
| Hematology |
| Chemistry |
| Soluble Serum Elements |
| African Horse Sickness |
| Foot and Mouth Disease |
| Rinderpest |
| Rift Valley Fever |
| Elephant herpes |
| Brucella Abortus |
| Leptospirosis (all 17 serovars) |
| Bluetongue |
| Johnes Disease |
| Parainfluenza-3 |
| Equine Infectious Anemia |
| Encephalomyocarditis |
| Bovine herpesvirus-1 (SN) |
| Bovine herpesvirus-2 (SN) |
| Bovine herpesvirus-4 (IPT) |
| Malignant catarrhal fever (IPT) |
| Equine Herpesvirus-1 |
| Equine Herpesvirus-2 |
| Equine Herpesvirus-3 |
| Equine Influenza virus |
| Equine Adenovirus |
| Equine Rhinovirus-1 |
| Equine Rhinovirus-2 |
| Toxicology Panel |
| Chlorinated hydrocarbons |
| PCBs |
| Heartwater (Cowdria ruminantium) |
| Mycobacterium Ag 85[b] |
| Pox Virus |

[a]Field Veterinary Program of the Wildlife Conservation Society; see: http://www.wcs.org/sw-high_tech_tools/wildlife-healthscience/fvp/168570/170390/fvp-elephants.

[b]The author (Mikota) recommends that the ELISA and MAPIA should also be included for TB.

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# 26 Reproductive System

Dennis Schmitt

## INTRODUCTION

The decline in free-ranging elephant populations is well documented and has been the result of complex factors throughout the preceding century. Understanding reproduction in elephants is important if we are to manage the free-ranging and captive elephants in our care effectively. An understanding of the anatomy and physiology of males and females is necessary in order to make informed decisions that will result in optimum care of the remaining elephants. Recent advances in our understanding of reproduction in elephants have resulted in successful births following artificial insemination in captive elephants, but the long-term success of elephants will depend on our ability to apply our knowledge of natural reproduction to the management of both free-ranging and captive elephants.

## REPRODUCTIVE ANATOMY OF THE FEMALE

The ovaries are relatively small in elephants and are approximately 7 × 5 × 2.5 cm in adults, composed of an inner medulla and outer cortex containing the follicles and corpora lutea (CL).[15] There are no large follicles or corpora lutea present until puberty. One dominant follicle normally ovulates at 15–25 mm diameter near the end of estrus. Typical corpora lutea derived from ovulation are large (>25 mm) and on the surface of the ovarian cortex; accessory corpora lutea seen during pregnancy are smaller and intracortical. The numbers of accessory corpora lutea increase during mid and late gestation and the total number can range up to 10 on each ovary, although 6–8 per animal is a reasonable average.[11]

The oviducts are approximately 10 cm long, and the ovaries are located relatively close to the tip of the uterine horn at the termination of the oviducts.

The uterus is 0.8–1.5 m long and is characterized by a short uterine body (5–10 cm) and relatively long uterine horns. Both horns run parallel in common connective tissue for 0.5–0.7 m to the uterine bifurcation, where each horn may be seen individually. The lumen of the uterine horn ranges in diameter from 12–45 mm.[15] The endometrium is composed of a homogenous mucosa, which becomes more prominent during estrus. The cervix is much like that found in horses, with longitudinal folds. The opening of the cervix protrudes into the vagina with an appearance like that of a rosebud, but the cervix has a short total length of approximately 15 cm.

The vagina has many longitudinal folds and measures approximately 30 × 15 × 10 cm. Unlike most other species the elephant penis does not physically penetrate the vagina for semen deposition.[1] During pregnancy thick vaginal mucus is present, which serves as a mechanical and infectious barrier. Nulliparous females have a hymen that does not rupture during mating. The vaginal os found in the hymen of nulliparous females is about 4 × 2 mm and is flanked by two blind pouches thought to be relics of Wolffian ducts. Postpartum, the opening to the vagina is approximately 2–3 cm in diameter and the blind pouches are not evident.[15]

The vestibule or urogenital canal in elephants is extremely long (1.0–1.4 m). It is a tubelike structure whose opening is between the hindlegs, runs vertically to just under the anus and curves horizontally at the cranial end in the caudal pelvis. The urethra and vagina open into the cranial portion of the urogenital canal, and a transurethral fold is present at the junction of the vagina and urethra. The clitoris is a relatively large structure cranial to the opening of the urogenital canal and is evident on relaxation of the vulva. The clitoris may help in directing the penis into the urogenital canal for copulation.

## REPRODUCTIVE PHYSIOLOGY OF THE NONPREGNANT COW

The endocrinology of the elephant estrous cycle has been well characterized, and the use of ultrasound to

evaluate corresponding changes during the estrous cycle has been investigated. See Chapters 27 and 28. A substantial number of elephants in captivity do not cycle normally. In North America about 28% of the African elephants and about 14% of the Asian elephants fail to cycle or have irregular estrous cycles.[4] Although most of the acyclic Asian elephants are over 30 years old, in Africans the prevalence is found among all age groups of adults.

## ARTIFICIAL INSEMINATION

Artificial insemination (AI) in elephants was attempted for several years without knowing the intricacies of the elephant estrous cycle. The timing of inseminations until the mid-90s were often off by several weeks from estrus. The use of radioimmunoassay to determine progesterone levels accurately on site was helpful, but the discovery of the unique double LH peak in elephants was instrumental in accurate timing of insemination.[5,23] See Figure 26.1. The first pregnancy from AI was produced by using the slight rise in progesterone prior to ovulation to determine the timing of insemination.[32] However, the ability to predict estrus accurately from the first nonovulatory LH peak enabled the scheduling of semen and an AI team 2–3 weeks prior to the AI instead of the day before. The use of ultrasonography is a component that allows the AI team to follow follicular development and predict impending ovulation, monitor the site of semen deposition (if needed), and confirm that ovulation has occurred.

A successful AI depends on providing viable semen during estrus, detecting the timing of estrus accurately, and delivering semen into the reproductive tract. Almost all the AIs performed to date have utilized fresh cooled extended semen inseminated the day of collec-

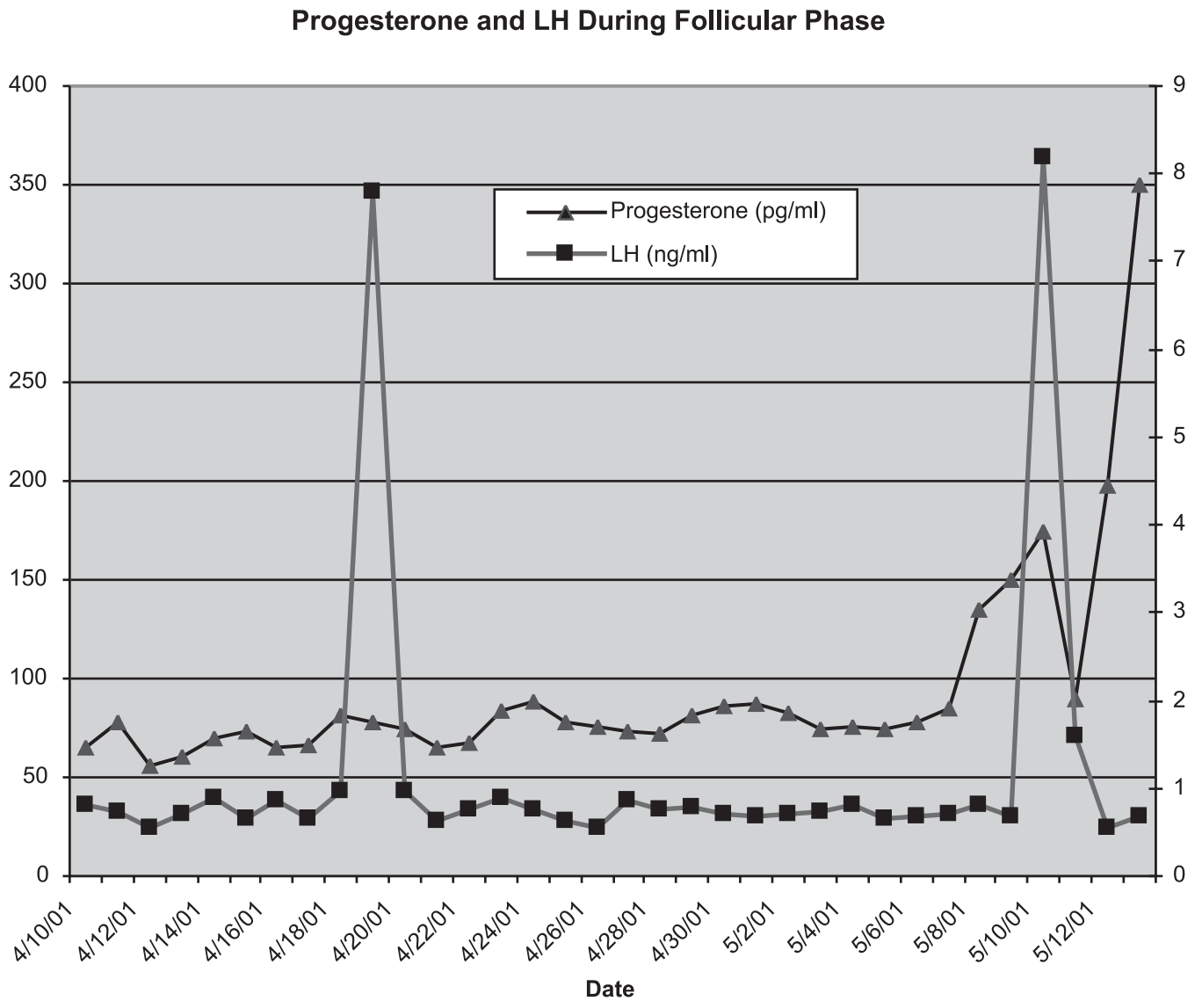


***Figure 26.1.*** *The unique double LH surges in elephants allow accurate timing for breeding or artificial insemination.*

tion. The coordination of the various facilities providing semen is an essential component. Usually 2–3 bulls are utilized to ensure that at least one of the semen donors will provide suitable samples for insemination. If multiple semen donors are utilized for the inseminations, parentage is determined by DNA analysis of the parents and offspring. The use of frozen semen for AI will provide assurance of viable semen for use at the proper time. Frozen semen has been used to produce one pregnancy and others will follow. Frozen semen provides a convenient source of semen for the facilities collecting semen and removes the uncertainty of whether semen will be available when the elephant is ready for insemination.

Nonsurgical insemination has been the predominant method utilized. It requires a well-trained and compliant elephant for insertion of a large bore cannula (similar in size to a large animal tracheal tube) into the lower urogenital tract.[3] Through the large bore cannula a 3 m endoscope is guided to the hymen (if the cow has not had a calf previously) and the opening into the vagina visualized. If the cow has previously had a calf, the endoscope may be used to visualize the cervix or in some cases the endoscope may be advanced up the uterine horn on the side of the ovulatory follicle. A small insemination cannula is then placed through the working channel of the endoscope and in the case of nulliparous cows the insemination cannula is threaded through the patent opening in the hymen and semen deposited into the vagina. In parous cows the cannula may be placed into the uterus through the cervix for semen deposition.

Surgical insemination may be useful in cows that do not tolerate urogenital manipulation well or cows that have urogenital polyps that hinder nonsurgical insemination. This technique is not as technically difficult, and does not require expensive equipment. A 1 cm incision is made just below the anus into the urogenital canal, after a surgical preparation of the surgical site has been completed and a local anesthetic utilized. A sterile disposable equine vaginal speculum with a small light source is used to visualize the hymen or cervix, and semen is deposited with the aid of an equine insemination pipette. Three to four sutures are placed at one-half skin thickness to close the surgical site. Aftercare of the incision site is required for 4–6 weeks until complete healing occurs and the sutures can be removed. Although this technique is less technically demanding, it requires more aftercare on the part of the elephant handlers and veterinary staff.

Artificial insemination is a tool that can be used with success in elephants that do not have access to a male for natural mating, or in elephants that are not compatible for natural mating. The success rate is between 30–40% for each attempt. This compares with an apparent success of 50–60% for natural breeding. However, the success of both AI and natural breeding require viable breeding animals. The importance of determining the reproductive status via endocrinology and ultrasonography is as important for natural breeding as it is for AI.

## PREGNANCY—PHYSIOLOGY AND DIAGNOSIS

Gestation in elephants is about 640–660 days (±14 days). Single births are predominant, although twins occur in about 1–2% of births. The age at first conception varies widely but is generally reported in free-ranging elephants at 10–12 years of age and in captivity may occur at an earlier age, perhaps due to nutritional differences resulting in early ages at puberty and higher growth rates.

Although there are relatively large amounts of data on the hormonal patterns during the estrous cycle of both species of elephants, relatively little information exists in African elephants, and most of the hormonal information during pregnancy is from Asian elephants. The information that is available from African elephants is from cross-sectional studies of elephants culled in the wild. Maintenance of elevated progesterone levels during early pregnancy suggests that conception results in the prolongation of corpora lutea function. Based on histological examination and progestin analysis of luteal extracts, CLs during pregnancy appear to be most active between 3 and 15 months of gestation.[19]

Considerable overlap of progestin levels between luteal nonpregnant and pregnant elephants make it difficult to use the absolute level of progestins as a pregnancy indicator, although some cows will have levels at 2–3 times their luteal phase values. In addition, use of two or three serum progesterone samples for pregnancy determination may be complicated by a transient fall in progesterone levels at 8–10 weeks postconception to near baseline levels before rising above normal cycling luteal levels. In the first half of pregnancy, progesterone is usually above normal luteal levels and falls slowly to near the levels found in the luteal phase of nonpregnant cows near parturition. The question of what supports CL function remains unanswered. Reliable evidence of a placental gonadotrophin has not been demonstrated.

Prolactin may serve as a luteotrophic factor in elephants, although this has not been demonstrated and its increased levels do not coincide with the increase in progesterone levels at 10–12 weeks gestation. Prolactin levels in pregnant elephants rise slowly beginning at 16 weeks of gestation and are significantly elevated after 24 weeks gestation. This allows the use of a single serum prolactin sample to confirm pregnancy in elephants that are pregnant 6 months or more.[6]

Diagnosis of pregnancy in elephants may be performed endocrinologically using prolactin or progesterone levels as noted above, but the development of ultrasonography methods for use in elephants has enabled pregnancy diagnosis and an evaluation of fetal viability. See Chapter 27.

Asian and African elephant placentas are nearly identical, with some minor differences. Implantation of the embryonic vesicle is near the base of one horn, with the yolk sac oriented mesometrial and the embryo antimesometrial within the horn. Implantation begins with equatorial replacement of endometrium and formation of an endotheiliochorial zonary placenta attachment. Placental scars are present from the placental attachment of previous pregnancies and may be used to estimate the number of total pregnancies at necropsy. Placental scars are also visible on ultrasound, but determining the number of pregnancies from ultrasonographic exam is extremely difficult after 2–3 pregnancies. A term elephant placenta is large and heavy, usually weighing between 15 and 24 kg (including free membranes). Aside from the size of the placenta two characteristics are usually noted: the presence of a brown to green girdle on the perimeters of the attachment of the zonary placenta and the "pustules" or verrucae found on the surface of the chorio-allantoic membrane. The primarily brown girdle on the perimeter of the attached placenta is thought to be a marginal hematoma, although the green color seen on the extreme edges of the attachment is yet to be adequately explained.[2] The free membranes of Asian placenta have a fine villous surface, but this is not true of the African elephant placenta. The allantoic pustules found in both African and Asians placentas are composed of loose connective tissue with numerous blood vessels.

The umbilical cord is about 100 cm (65–170 cm) long, but it does not allow delivery of the calf without detachment of the cord from the fetus. The length of the tract from the uterus until expulsion of the calf from the vulva results in detachment before expulsion. Detachment occurs at the surface of the fetal abdomen, and the umbilical vessels retract into the abdominal cavity with a short external sheath remaining externally. The umbilical cord has three blood vessels at its attachment to the fetus and the allantoic duct, which delivers fetal urine into the allantoic sac.

## MANAGEMENT OF THE PREGNANT COW

The approximately 22-month gestation may be viewed as having three different phases. The first phase is embryonic vesicle development, which is thought to contain an embryonic diapause during the first 9 weeks of gestation. From 10–18 weeks gestation there is a rapid growth of the fetus and fetal organs. It is during this phase that the fetus can first be identified visually as an elephant by the distinctive trunk. During much of the remainder of gestation there is continuing slow growth of the fetus. At 1 year of gestation the fetus is approximately 30–35 cm in height and length. During the last few months of gestation, the fetus gains weight more rapidly until parturition, when the calf usually weighs in excess of 100 kg (220 lb) and is about 80–90 cm in height and length. The first visual indication of pregnancy is mammary development. There may be slight swelling of the breasts beginning at 6 months, but usually a change is detected by palpation of the breasts for an increase in glandular firmness and size deep in the tissue. As the pregnancy proceeds the mammary glands become more obvious.

Until late pregnancy there is little demand for nutrients for growth of the fetus. Additional weight gain from pregnancy through the first year is minimal because the fetus, placenta, and fetal fluids total approximately 30 kg. However, in captivity pregnant elephants often gain significant weight during this period. Overweight elephants are a problem in captivity and pregnant elephants that are overweight are at increased risk for dystocia and stillbirth. Pregnant elephants need to be monitored for weight gain and involved in an active exercise program.

Regular monitoring of serum chemistries and a CBC is recommended throughout pregnancy in addition to weekly progesterone monitoring. As the cow nears parturition, progesterone monitoring is increased to provide warning of impending birth so that adequate preparations can be made and assistance provided if necessary. Total weight gain during pregnancy should be less than 250 kg (550 lb) in a mature cow. It is not until nursing begins that significant nutrients are required by the dam to provide milk for the calf. A well-balanced ration should be provided throughout pregnancy for normal fetal development while weight gain is being monitored.

## PARTURITION

Serum samples for progesterone assay are recommended every other day from week 89 to 91 of gestation. Samples can be assayed two to three times weekly to monitor serum progesterone levels. From week 91 (637 days) daily samples are recommended and a laboratory that can run the samples daily utilized. Many laboratories utilize progesterone assays that do not have the sensitivity needed to monitor progesterone in elephants near term. The level of sensitivity needed for the assay is 0.05 ng/ml (50 pg/ml).

Impending parturition is indicated in most elephants by a precipitous fall in progesterone to baseline levels for that elephant, usually less than 0.10 to 0.15 ng/ml, over a period of 1–2 days. Progesterone usually falls 50% in a 12–24-hour period, and then drops to baseline levels in the next 12–24-hour period. Birth usually occurs 2–5 days following the return of progesterone to baseline levels, although normal healthy calves have been born 7–12 days following the decline in progesterone levels. There have been a couple of exceptions where parturition occurred when progesterone was above baseline levels.

Changes observed in the cow near parturition include increased beating of the vulva with the tail, fre-

quent production of small fecal boluses and small quantities of urine, sudden "freezing" during movement, stretching, abdominal discomfort, refusal of normal rations, and emission of milk from the mammary glands.

Two of the most relevant events for impending parturition are the passing of the mucus plug and rupture of the fetal membranes. Passing of the mucus plug is observed in about 50% of births. It is a thick opaque viscous plug that may not be seen if stepped on or covered with bedding, feces, etc. Usually parturition occurs within 24 hours of the mucus plug being passed. Rupture of the fetal membranes occurs within 2 hours prior to birth in most cases.[30] Milk from the mammary gland may be present for varying times prior to parturition. A change in the concentration of calcium has been utilized in those cows with milk present prior to birth. Many cows do not produce milk in the mammary glands until a few hours prior to parturition, and some will not have milk present until parturition occurs. Ventral edema is not unusual near the time of parturition and following birth of the calf.

Preparations for calving include having milk replacer available in case it's needed. See Chapter 16 for details.

An area for the birth to occur should have all obstacles removed and be calf-proofed so the calf cannot escape from the area. Check all restraints and fastening points. An absorbent material such as bran or sawdust should be used to soak up the amniotic fluids to provide better footing for the elephants and the keepers on a wet concrete floor.

Supplies to have available include two to three pairs of soccer gloves to get a better grip on the wet slippery calf when needed, and a soft woven cotton girth strap may be used to support an unsteady calf and to restrain sudden movement until the calf is able to stand and walk well on its own. The girth strap may be placed between its forelimbs from either side to enable keepers to remove the calf from the cow's side rapidly if the cow becomes excited or aggressive.

Parturition begins when the calf is positioned in the uterus for delivery (stage 1). Little discomfort is usually seen in elephants other than the subtle "freezes" and abdominal discomfort. Active labor (stage 2) usually is not seen until the cervix is dilated and the calf presented into the birth canal, although some cows will exhibit obvious labor in the process of dilating the cervix and expelling the calf into the birth canal. If rupture of the fetal membranes occurred over 2 hours previously and active labor has not been seen, veterinary intervention should be initiated. In the absence of active labor, if a mucus plug or fetal membrane rupture is not observed, the first indication of impending birth may be the appearance of a bulge under the tail. This bulge may be the unruptured chorio-allantoic membranes or may be the calf being delivered in the amniotic sac.

Anterior and posterior presentations are normal with the feet extended. The presentation of the calf may be determined by the position of the toenails. In anterior presentations the toenails are dorsal; in posterior presentations the toenails are ventral. The feet and nails of the calf may be felt through the rectal wall as the calf is in the pelvic canal. Normally the calf is expelled from the pelvic portion of the birth canal and turns the corner down the urogenital canal and the feet and amniotic sac are seen protruding from the vulva. The calf will suspend under the abdomen until a significant portion of the calf's body is external to the canal. It then drops to the ground, and if the cow is experienced the calf will be moved by the cow to stimulate the calf to move. Movement of the calf will often break the amniotic sac or it may be broken by the dam. The cow will try to assist the calf to right itself, and she will check on the status of the calf by bending down to assess the calf's condition visually. Inexperienced cows may perform the same actions, but be overly aggressive and anxious and harm the calf. In older inexperienced cows the calves have been severely injured and in some cases death of the calf results. The experience of the staff attending the birth is critical in assessing the ability of the cow to nurture its calf immediately after birth. To increase the survival of newborn calves it is recommended that cows in labor be restrained so that they are able to have normal labor, and the calf can be safely moved to an area in the view of the dam, but out of her reach until the calf is able to walk and is ready to nurse. This gives the staff time to assess the intent of the dam and decide when the calf can be allowed to interact with the dam. If the dam is accepting and exhibiting appropriate maternal behavior, the calf is given restricted access to the dam to begin nursing.

Stage 3 of parturition is expulsion of the placenta and uterine involution. Expulsion of the placenta usually occurs within 10 hours of birth. Retained placenta has occurred in a few cases and may take 2–4 weeks to be expelled although most are expelled much sooner. Postpartum, the cow will pass dark brown lochia for several days as uterine involution begins. Complete uterine involution probably takes several weeks.

In captive African females there appears to be about a 2-year delay in return to estrus; in Asian elephants several pregnancies have occurred around 8 months following parturition.

## DYSTOCIA

Veterinary intervention depends on the personal experience of the facility and the attending veterinarian. The balance between leaving the dam undisturbed and needing to evaluate progress in delivery of a viable calf is delicate. Elephants appear to be able to stop active labor if disturbed except during the final expulsion of the fetus, much like horses. Veterinary supplies to have available include plenty of lubricant, an ultrasound if available, oxytocin, estrogen, tamed iodine, lidocaine,

xylazine, butorphanol, detomidine, atipamezole, doxapram, and oxygen, in addition to stethoscope, thermometer, etc.

When to start veterinary intervention is still to be determined, but earlier intervention may be beneficial. Ultrasonography to determine fetal viability, cervical dilation, or presentation of the calf in the birth canal is essential. Familiarity with the use of ultrasound in elephants is needed to use it effectively for parturition management. See Chapter 27. Transverse and longitudinal views of the cervix and vagina give accurate information on cervical dilation and the presentation of the calf. Until the cervix is dilated and the calf is positioned for delivery, no veterinary intervention is needed except to continue to monitor the process.

After the calf is positioned in the birth canal and the cervix is dilated, frequent monitoring for progress may be needed if no outward indications of labor are seen. If the membranes have ruptured over 2 hours previously and the calf is not making progress in the birth canal, veterinary intervention is warranted; intervention is also warranted if it has been more than 120 hours since progesterone fell to baseline but the cervix is partially dilated. Elephants are sensitive to injectable oxytocin and administration of 50–60 IU of oxytocin SC or IM is effective in initiating active labor. However, some cows may be stimulated into active labor by rectal massage with both arms in the pelvic area.[30] To stimulate further relaxation of the cervix, administration of estrogens may be considered. In other species, estrogen is utilized to potentiate oxytocin receptors in the uterus; this may be helpful in elephants that don't respond to oxytocin after a long labor. If the response to oxytocin results in only one or two strong contractions and labor stops, the position and size of the calf should be evaluated for its ability to be expelled naturally. The normal response to 50–60 IU of oxytocin is a gradual increase in contractions that begin 5–10 minutes after administration and lasts for 20–30 minutes. If the contractions continue beyond 30 minutes, there is no need to continue to administer oxytocin. If the contractions stop and do not reappear, evaluate the progress of the calf down the birth canal. If progress has been made, continue to use 50–60 IU oxytocin; if no progress has occurred, the dose may be increased to 100 IU and the position of the calf in the birth canal reevaluated. The 100 IU oxytocin should be readministered as long as progress toward delivery is occurring.

If no progress in delivery of the calf can be achieved using oxytocin, a decision must be made on the next level of intervention. It is best if the various scenarios are discussed prior to parturition and tentative decisions made about which interventions will or will not be attempted, should the need arise. The universal failure of all cesarian sections to date [8,9,27] in elephants has resulted in the decision not to attempt surgery in most cases, if any. Currently the options being utilized are vestibulotomy and extraction of the calf (through traction or fetotomy) or leaving the calf in utero to be expelled at a later date. Both have their risks and potential consequences.

Vestibulotomy is a surgical procedure through which the vertical part of the urogenital tract is incised and direct access to the calf is provided.[24,25,29] Vestibulotomy is contraindicated if the fetus is not accessible in the birth canal and confirmed by ultrasonography and rectal palpation. The incision into the urogenital canal is made after local anesthesia is administered and a 10 cm skin incision made vertically just under the anal fold. Introduction of a surgical guide into the urogenital canal facilitates a quick incision into the canal through the skin incision without risk of cutting the large blood vessels lining the cranial side of the urogenital canal. The initial incision is then extended to an adequate size to allow manipulation of the calf and its extraction through the incision. Provision of adequate lubricant is essential in manipulation of the fetus through the birth canal, and extraction of the calf should be performed as if it were a foal, only much larger. Downward traction should be applied to one leg at a time to "walk" the fetus through the pelvis. If the calf can be rotated 90° it may be helpful in delivery of a relatively oversized fetus. If the fetus cannot be delivered by traction, a fetotomy will need to be performed. Delivery of the calf through traction or fetotomy is a difficult procedure and adequate help should be available to be successful.[17,18]

Leaving the fetus in the birth canal to be expelled later is not a recommended veterinary procedure in other species. There have been reports of elephants carrying calves past term and expelling calves after several months to a few years and subsequently becoming pregnant and successfully calving. If the fetus is not available to extract in the pelvic canal after unsuccessful attempts to expel the calf, the only real alternative is to leave the calf in the uterus and wait for the dam to go into labor and expel the calf later. If the calf can be reached, a decision to leave the calf in the uterus becomes more difficult. There have been cases where the calf was available but was determined to be too large to deliver through traction and was left in the birth canal. The calf regressed into the uterus and was expelled a few months later or was able to be extracted via vestibulotomy. In other cases the calf was left in place, and uterine rupture or degeneration occurred subsequent to the labor and the dam was lost.

Dystocia in elephants is not an easy dilemma to solve.[9,10] All options must be weighed, and an informed decision made that results in the best outcome possible for the dam, calf, and institution.

## REPRODUCTIVE ANATOMY OF THE MALE

The testes of the elephant are located intraabdominally and hang from the dorsal wall of the abdominal cavity

medial and slightly posterior to the kidneys. Elephant testes are unique because there is no pampiniform plexus to assist in cooling the testes below body temperature. However, normal body temperature in elephants is 34–36°C which is about the temperature at which the testes of scrotal mammals are maintained. A single testicular artery penetrates the testis and radiates smaller arteries from the hilus of the testis. This is in contrast to mammals with scrotal testes where the testicular arteries are present on the surface of the testis and penetrate the parenchyma from the surface after further cooling of blood in the scrotum. The testes are ovoid in shape and vary in size during development of maturity. The immature testes range from 2 cm (newborn) to ~9 cm (prepubertal). The internal structure of neonatal and subadult males contain a central major blood vessel embedded in the meadiastinum testis. Sperm-rich samples were collected in one study only if they had testicular diameter of more than 10 cm diameter.[16] Hildebrandt also reported that testes of subordinate inactive breeding bulls have smaller testes than the dominant breeding bulls.[16]

The measurement of testosterone in peripheral blood did not reflect the differences in size and echogenicity between the small inactive and large active testes, and it may not be useful in determining the reproductive status of a bull elephant. A mature breeding bull's testis may weigh up to three kg and measure 17 cm or more in its largest diameter. The ovoid shape is defined by a testis that measured 17.5 × 15.0 × 11.5 cm. The seminiferous tubules range in diameter from 124–232 microns and increase in diameter with age. The numbers of Leydig cells are also quite variable and may occur in clusters.[26]

The epididymides of the elephant are not adhered to the surface of the testes as in scrotal mammals. The epididymis is a long cord (150–200 cm, depending on the age and size of the bull) highly convoluted duct with three anatomically distinct regions; head, isthmus, and tail.[22] The vas deferens is relatively short and coiled.

The ampulla gland is the widened terminal portion of the vas deferens, glandular in nature and considered one of the accessory sex glands. Ampullae glands are located dorsal to the neck of the bladder as it enters the urethra. It serves as a storage area for sperm until ejaculation. On ultrasound exam the amount of filling of the ampullae can be utilized to predict the success of semen collection. Well-expanded ampullae have a diameter of ~5 cm and are cone shaped, with length of 6–8 cm.

The seminal vesicles are the largest accessory sex glands in an actively breeding bull and may contain up to 1.5 l of fluid each. Fluid in the seminal vesicles make up most of the volume of a normal ejaculate and it is high in fructose. There is an outer muscular and inner mucosal layer that can be clearly seen on ultrasound. Seminal vesicles are each located lateral to the ampulla and dorsolateral to the bladder. In a healthy bull, seminal vesicles combine to provide 25–150 ml of fluid per ejaculation, up to five times per day, during natural breeding.[16]

Prostate glands are paired structures, with each half having three lobes joined by a bridge of tissue. It is located above the pelvic urethra caudal to the ampullae. Prostate glands in Africans and Asian are different in their size and composition. African elephant prostates are larger (up to 5 cm diameter on each side) and contain irregular-shaped internal cavities with prominent glandular tissue in each of the three lobes. Asian elephants have 2 cm diameter prostate glands, and the tissue appears fairly homogeneous.

The last of the accessory sex glands found in the elephant are the bulbourethral glands. These 10–12 cm paired glands are found immediately caudal to the root of the penis and the dorsal lateral aspect of the urethra close to the ischial arch. The glands are tubulalveolar and are covered by a muscular capsule. The fluid found in the bulbourethral glands is a thick clear fluid and may be involved in the cleansing of the urethra prior to ejaculation and in the lubrication of the vulvar area of the cow to help with intromission of the penis into the vestibule.

The penis of an adult elephant is heavily vascularized and has been described as ~100 cm long and 16–20 cm in diameter and may weigh over 27 kg.[26] There is some discussion of whether they have a true prepuce or glans penis. The erect elephant penis has an S-shaped curve and the paired levator penis muscles are large. The erect elephant penis is very mobile and allows the penis to move independent of the pelvis. During mating, very little pelvic thrusting or movement by the male is necessary for intromission and ejaculation.

## REPRODUCTIVE EVALUATION OF THE BULL

The objective of a reproductive evaluation of a bull is to assess his breeding potential in a natural breeding environment. The limitation of a reproductive evaluation of a bull elephant is that access to a normal ejaculate for semen evaluation is not always possible. The three methods used in other species are utilized in elephants, but they are limited by the access to normal breeding activity. In cattle and horses an artificial vagina (AV) is used with a phantom or animal mounted for normal breeding and the penis deflected into an artificial vagina. The use of an AV in elephants is a trained behavior and the bull restrained with all four feet on the ground. A partially erect penis is inserted into the AV and an ejaculate collected. In a collection of a bull trained to use an AV, no difference in the size of the seminal vesicles was noted pre- and postejaculation.

The sample was a highly concentrated sample similar to that found when rectal massage of the urethra is used to stimulate penile protrusion and massage of the ampulla when an ejaculatory response is noted.[31] Manual

massage of the urethra and stimulation of the ampulla glands result in a 30 ml average ejaculate in about 60–80% of the bulls collected. Motility varies greatly, from no motility to excellent motility. This may be due to the failure to collect a complete ejaculate seen with natural breeding or electroejaculation.[21]

Sperm cells in elephants are smaller than other domestic animals and contain dense bodies not seen in other species. African elephant semen appears to tolerate rather simple extenders and may be maintained and cooled for shipment in TL-Hepes media for study at a central laboratory or for artificial insemination.

The sperm membranes in Africans have relatively more unsaturated, long-chained fatty acids than found in Asian elephant sperm membranes.[34] Cryopreservation of African elephant semen is somewhat more successful than with Asian. The first successful pregnancy from AI with frozen semen in an African elephant was recently announced using semen frozen 1 year earlier.

Successful freezing and thawing of Asian elephant semen has several obstacles to overcome. Perhaps as a result of the differences in sperm membrane composition, Asian elephant semen appears to be susceptible to spontaneous acrosome degeneration, unless a complex extender is utilized immediately to prevent deterioration of acrosome integrity. Currently low levels of dimethylsulfoxide (DMSO) are used successfully to allow successful artificial insemination in Asian elephants with fresh semen utilized the same day as collected. Frozen Asian elephant semen has not been used successfully in an artificial insemination, to date. However, a study performed in Thailand suggests that a relatively simple extender with glycerol may be used to freeze Asian elephant semen successfully with adequate motility upon thawing.[35]

Ultrastructurally, elephant spermatozoa are different from domestic ruminants in several respects. The acrosomal ridge is difficult to visualize with light microscopy, but may be stained with various acrosomal stains for acrosomal integrity and morphological changes. The equitorial segment extends more distally on the ventral side of the head. In the neck region, dense material is located near the top of the mitochondria; the function of the dense spherical masses is not known. The dimensions of Asian elephant sperm have been well described and have a head about 7.8 µm long and 4.7 µm at its widest. The midpiece length is about 11.1 µm and the tail about 47.3 µm long, for a total length of about 66 µm.[12]

## MUSTH MANAGEMENT

Musth has been well documented in Asian elephants for hundreds of years and has recently been recognized as a phenomenon in Africans. See the section on olfaction in Chapter 31.

Chemical control of musth has included the use of medications that alter the release of LH from the pituitary. One recent publication summarized the use of Leuprolide acetate, a gonadotrophin releasing hormone (GnRH) agonist. Injections were administered during early or premusth, and a significant increase in testosterone levels resulted for 10–14 days with a subsequent decline due to down regulation. The results suggested the leuprolide could be a suitable alternative for controlling or preventing musth, although permanent reproductive effects may occur.[7] Newer GnRH antagonists may have promise in controlling musth behavior in bulls when needed. But we must remember it is a natural phenomenon in healthy adult bulls, and the need is to manage bulls in a way that is safe for them and those around them.

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# 27 Reproductive and Diagnostic Ultrasonography

Thomas B. Hildebrandt

## INTRODUCTION

Ultrasonography is mainly a reflection imaging technique in contrast to radiography as a transmission imaging technique or magnetic resonance imaging (MRI) as an emission imaging technique. Diagnostic ultrasound (2.0 MHz to 50 MHz) produces a cross-sectional anatomical picture of how sound waves reflect, refract, and are absorbed by different tissue. Ultrasound (US) is generally noninvasive, except for the applications listed in Figure 27.1. Ultrasonic energy waves can cause minimal bioeffects such as mild tissue warming, especially if color-flow Doppler Mode is used for extended periods of time (>30 min). However, there has been no report of patient injury or US-induced discomfort when examinations were performed under the international US safety guidelines. The necessity for animal handling and, potentially, sedation may add invasive components to the application of US in nondomestic species.

In 1991, Adams[1] described the first use of US in rhinoceroses and elephants. Special accessories and protocols were developed over the next few years to increase the efficacy of US in these megavertebrates. Since 1993, the Berlin team has performed over 3000 individual US examinations in approximately 350 Asian and African elephants in different human management settings and in the wild.

The use of ultrasonography offers new opportunities in elephants to evaluate parts of internal organ systems and joints that are inaccessible by other means (e.g., conventional/digital radiography, magnetic resonance, endo/arthroscopy) due to the enormous size of the elephant and the location and dimension of the organs. In contrast to other imaging procedures, clinical ultrasonography has several advantages: 1) it is safe due to minimal bioeffects and therefore repeatable; 2) it provides real-time information; 3) it generates high-resolution characterization of soft tissue and bone surface as well as morphometrics of organs, implants, or other foreign bodies; 4) it produces sectional images and 3D reconstructions of tissues and organ structures; 5) it permits examining motion and direction (heartbeat, vascular flow, fetal movement); 6) it operates economically and efficiently; 7) it facilitates documenting and preserving data on storable media; and 8) it is portable and compatible with zoo and field conditions.

Approximately 15% of all US applications in elephants are performed transcutaneously, compared to 85% transrectal applications, because of the anatomy of the integument and the restricted penetration depth of commercially available US systems (maximum average depth = 220 mm). The transcutaneous approach is predominantly used for obstetrics (late-term pregnancy), orthopedics, cardiology, dentistry, ophthalmology, and for the visualization of cranial abdominal organs and mammary gland development and function.

Transrectal US is used for imaging the urogenital system, rectal wall, intestinal loops, peritoneal-abdominal cavity, and early to midterm pregnancies. A variety of transducers with frequencies ranging from 2.0 to 10.0 MHz may be used with transrectal US, whereas transcutaneous US may be performed only with a low-frequency probe (e.g., 4–2 MHz). Transrectal US provides greater detail, although a higher level of patient preparation is needed and specific customized accessories are required.

## PATIENT PREPARATION FOR ADULT ELEPHANTS

Note that certain modifications are necessary for US use in adolescent elephants.

### Transcutaneous Ultrasound

Transcutaneous US may be performed in free contact (see Fig. 27.2), in protected contact using a chute or a training wall, or in immobilized elephants in lateral recumbency. With full immobilization the admission is limited to only one side. As a precaution in unanes-

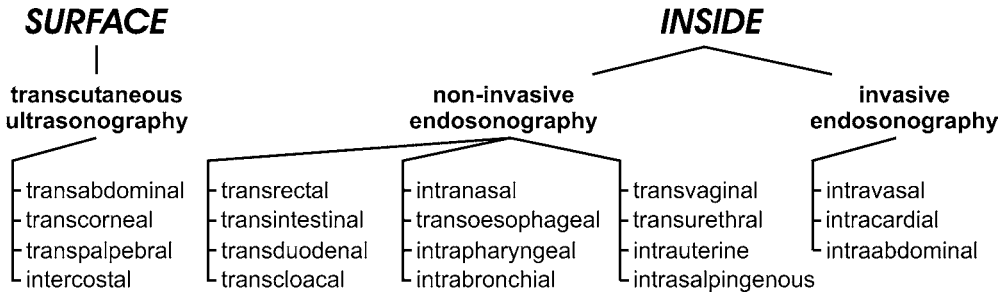


***Figure 27.1.*** *Overview of the ultrasound (US) applications in mammals. Some approaches are not practicable for elephants.*

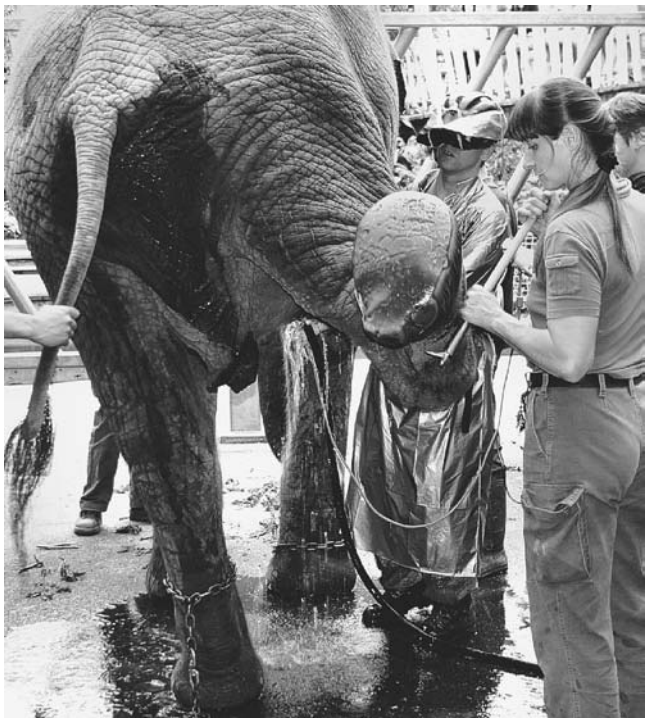

***Figure 27.2.*** *Transcutaneous US application for pregnancy monitoring that is relevant in the second half of gestation. The running water from a hose improves the coupling between the US transducer and the highly folded skin. Note that there are three keepers (head, tail, leg) present to guarantee a safe examination under direct contact conditions. The leg lifting improves the accessibility to the abdomen. In general, the entire procedure should not exceed more than 20 minutes and should be combined with continuous food rewards in order to keep such operations positive for the elephant.*

thetized animals, there should always be at least one elephant keeper at the animal's head and one keeper at the tail during the entire US investigation.

Certain anatomical features of the integument must be compensated for in order to achieve good image quality with transcutaneous US. The normal epidermal layer of an elephant is only 5 mm thick; however, it is deeply folded, creating the characteristic wrinkles of the elephant skin. These folds trap air and sand, which hinder US beam penetration. In captive situations, elephants may develop pathologic hyperkeratosis, which completely eliminates the possibility of transcutaneous US. The subdermis may be 10–15 mm thick and in combination with the epidermis it is strongly absorptive, reducing the US beam energy. Therefore, intensive cleaning and removal of foreign particles from the skin is a prerequisite for transcutaneous US examination. High-pressure washing systems used in some elephant barns may be helpful.

Running water is applied directly to the transducer field during scanning to minimize interference by trapped air and replaces the traditional US gel, which may exacerbate the problem. The water should be free of bubbles.

In adolescents, the integument is less folded and thinner and therefore the negative effect on the image quality is less dramatic. This combined with the smaller size allows broader application of transcutaneous US.

## Transrectal Ultrasound

Most assessments are performed in free contact with the elephant standing or in lateral recumbency without the use of tranquilizers, anesthetics, or restrictive devices. There have been no remarkable differences in image quality or accessibility to the internal urogenital tract among examinations performed in different settings, such as free contact, protected contact (use of restraint chute), or no contact—including animals from the wild (under anesthesia).

An important prerequisite for good-quality imaging is thorough cleaning of the distal 2.5 m of rectum (see Fig. 27.3). Feces are removed manually and the rectum irrigated with lukewarm water from a hose with a smooth tip (10–20 mm diameter) at a flow rate of 10–20 liters per min. Lubricant is used in addition to commercial US gel to overcome the anal tone painlessly. In nervous individuals, multiple enemas may be required during the examination. Additional lubricant and a reduced flow rate may be needed to avoid damage or pain in elephants with strong anal contraction. Most elephants respond within a few minutes to rectal manipulation and the enema by lifting their tails (Fig. 27.4).

Some older elephants, mainly African, develop rectal polyps in the region of the mucocutaneous junction, which could be pain sensitive during rectal palpation. The application of xylocaine gel normally used for intu-

***Figure 27.3.*** *The application of a rectal enema in preparation for the following transrectal US examination. It is important that the tip of the hose be smooth and the water lukewarm with a maximum flow rate of 20 liters per minute. Note that there are three keepers (head, tail, side) for safety reasons.*

***Figure 27.4.*** *A transrectal US examination in a female elephant in direct contact. The examiner performing the transrectal exams guides the ultrasound probe with the help of the video glasses (built into the helmet), and the other examiner assists by operating the US machine.*

bation helps relieve this discomfort. As a safety precaution in unanesthetized animals, there should always be at least one elephant keeper at the animal's head and one keeper at the tail during the entire US procedure. Positive reinforcement is provided by feeding favored foods. Discomfort is usually demonstrated by the animal refusing treats. By following these general guidelines, daily rectal examinations may be performed over several weeks without any behavioral problems or rectal lesions. In some cases, the US examinations may be-

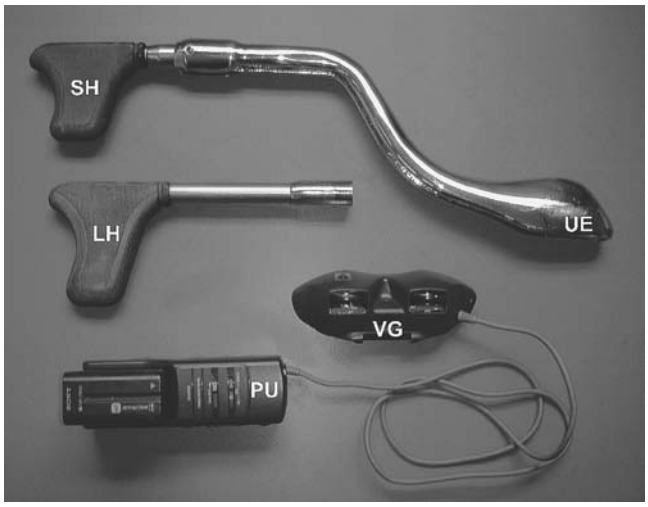


***Figure 27.5.*** *The upper part of the image shows the US probe extension (UE) for imaging the cranial parts of the urogenital systems (see Table 27.1), optionally equipped with a short handle (SH) or a long handle (LH). The lower part of the image shows the digital video glasses (VG), which are connected with the processor unit (PU). For protection and better handling, these video glasses are built into a customized bicycle helmet (seen in Fig. 27.4), allowing the examiner performing the rectal US exam to have direct visualization of the US images independent from the US machine.*

come part of the daily routine, similar to bathing, foot care, and blood sampling.

Transrectal US examination with handheld transducers is possible after about 3 years of age. Before this age, the rectal diameter is too narrow for safe manipulation and examinations may result in rectal injury. Young elephants are more active which adds an additional risk of injury during manipulation. Fully immobilized elephants under 3 years of age may be examined using a bear probe extension[12] and a standard horse rectal probe, which avoids the need to insert a hand or arm. The customized elephant probe extension (see Fig. 27.5) is not recommended for use in nervous elephants, in individuals under the age of 6 unless immobilized, or in the late stages of pregnancy.

## ULTRASOUND SYSTEMS AND ACCESSORIES

There are a plethora of US systems commercially available; however, the price and the special features needed for application to elephants drastically limits the options. US machines equipped with mechanical scan heads are not recommended. They are more fragile, and elephants react strongly to the vibrations. The ideal system for basic US in elephants is a portable, battery-powered, real-time B-mode, with or without the color Doppler option, equipped with at least one low-frequency convex (3.5 MHz), one middle-frequency microconvex (5.0 MHz), and one high-frequency linear (7.5 and 10.0 MHz) transducer. Newer systems offer

**Table 27.1.** Ultrasonographic Imaging of the Urogenital Organs Compared to the Applied Transducers

| | | | 3.5 MHz | | | 7.5 MHz | | | 10.0 MHz* | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Organ | | Adapter | Overview | Details | Surface | Overview | Details | Surface | Overview | Details | Surface |
| Male | Testes | Yes | +++ | +++ | ++ | — | ++ | +++ | — | — | — |
| | Epidymides | Yes | + | — | — | ++ | ++ | ++ | — | — | — |
| | Ductus deferentes (cranial portion) | Yes | — | — | — | — | — | — | — | — | — |
| | Ductus deferentes (caudal portion) | No | ++ | + | + | +++ | ++ | ++ | ++ | +++ | +++ |
| | Ampullae | No | +++ | ++ | + | +++ | ++ | ++ | ++ | +++ | +++ |
| | Seminal vesicles | No | +++ | ++ | ++ | ++ | +++ | ++ | + | ++ | +++ |
| | Prostate | No | +++ | ++ | + | +++ | +++ | ++ | ++ | ++ | +++ |
| | Bulbourethral glands | No | — | — | — | — | — | — | — | — | — |
| Female | Ovaries | Yes | ++ | + | — | +++ | +++ | +++ | — | — | — |
| | Uterine horns | Yes | +++ | ++ | + | +++ | +++ | +++ | — | — | — |
| | Uterine body | No | +++ | +++ | ++ | ++ | + | ++ | — | + | +++ |
| | Cervix | No | +++ | +++ | ++ | ++ | ++ | ++ | + | ++ | +++ |
| | Vagina | No | +++ | +++ | ++ | ++ | +++ | ++ | + | ++ | +++ |
| | Vestibule (cranial portion) | No | +++ | +++ | ++ | ++ | +++ | ++ | — | ++ | +++ |
| General | Kidneys | Yes | +++ | +++ | ++ | + | ++ | +++ | — | — | — |
| | Urinary bladder | No | +++ | ++ | + | + | +++ | ++ | — | ++ | +++ |
| | Ureters (cranial portion) | Yes | — | — | — | — | — | — | - | - | - |
| | Ureters (caudal portion) | No | +++ | ++ | + | ++ | +++ | ++ | - | ++ | +++ |
| | Urethra | No | +++ | +++ | ++ | ++ | ++ | ++ | + | ++ | +++ |
| +++ | = excellent | | | | | | | | | | |
| ++ | = good | | | | | | | | | | |
| + | = insufficient | | | | | | | | | | |
| - | = impossible | | | | | | | | | | |

*= this type of transducer did not fit in an adapter.

broad frequency transducers, which provide better image quality. For example, the classic 3.5 MHz is comparable to the new 4-2 MHz transducer. The scanners should be silicone sealed before use with water and in rectal examinations. Electrical safety must be considered at all times and especially in the presence of running water. Specific modifications—such as using waterproof cable connections and, perhaps, waterproof covers for the US system—are necessary to prevent shock of the examiner or the elephant. Table 27.1 shows the efficiency of three different probe frequencies applied with and without probe extension in the ultrasonographic imaging of urogenital organs in elephants. The system should be durable for use outside a protected examination room (Fig. 27.6). For specific research purposes, high-end stationary systems, such as 3D or 4D US systems (Fig. 27.7), are feasible. The use of these specialized systems requires extensively trained animals and special operating skill as well as special facilities (dust-free and dry).

Accessories for elephants include helmet with digital video glasses cable extension, video cable connection, digital portable battery-powered VCR, and varying probe extensions (Fig. 27.5). Not all are commercially available; the probe extensions and the video helmet have been custom designed for US application in elephants.

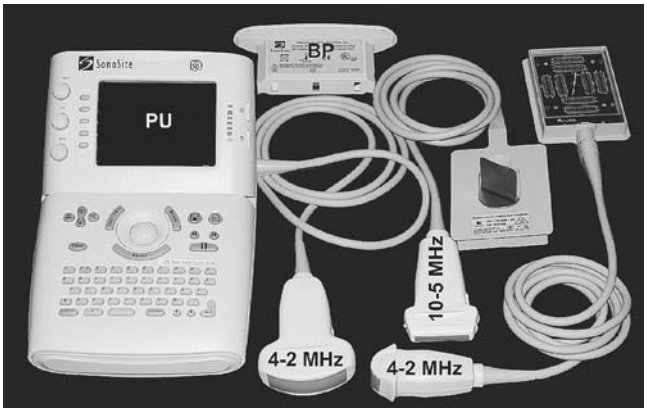


***Figure 27.6.*** *Shown is one commercial portable US system (PU), which can be used for elephant assessments. The PU is equipped with one low-frequency (4-2 MHz) convex transducer, one low-frequency (4-2 MHz) micro-convex transducer, and one high-frequency (10-5 MHz) linear transducer.*

## TRANSCUTANEOUS ULTRASOUND IN ELEPHANTS

A low-frequency scanner (4-2 MHz) may be used to image all structures from the skin surface to a depth of 220 mm. This includes the intact integument and subcutaneous le-

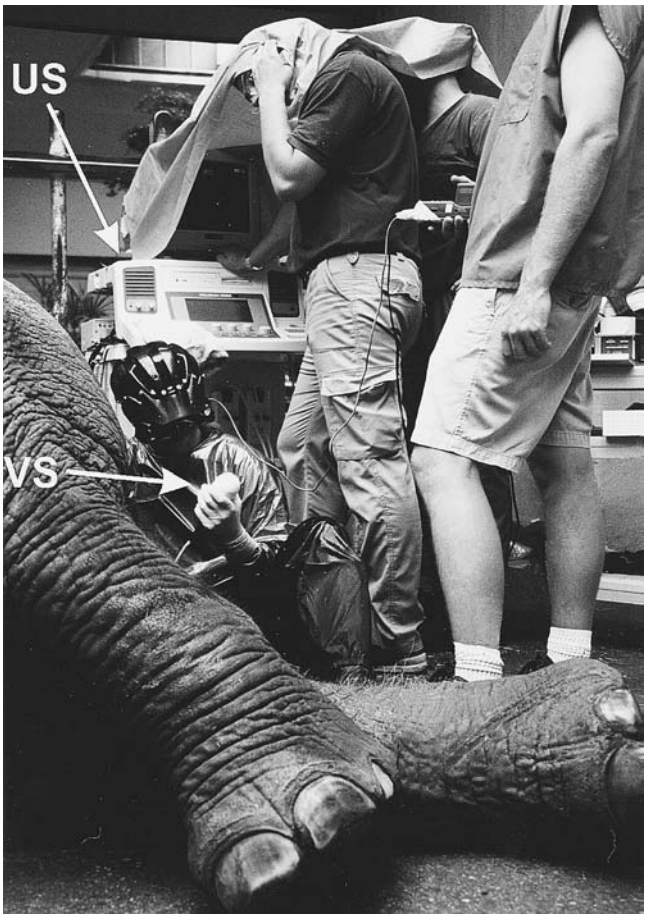


***Figure 27.7.*** *Setup during an US examination with a 3D system. The examination can be performed only in lateral recumbency—the transducer cables in these high-end machines are shorter because of the near-ground cable connection location. The specific volume scanner (VS) is larger and causes vibrations during the scanning process. This can cause slight discomfort for the mother as well as for the fetus. In general, this procedure should be applied only in well-trained elephants and used for research purposes exclusively.*

sions such as abscesses. US may identify the etiology of a lesion and help determine the optimal treatment.

US may also differentiate between lesions that superficially appear similar (abscesses, hematomas, tumors, edema) by characterizing their internal composition. Edema is a common finding in periparturitant females, females with retained calves, and females with late stage tuberculosis and severe generalized infections such as pox virus (indigenous only for Europe).

Elephants with tusks may develop minifractures resulting in free bone chips. These isolated bone fragments may cause dramatic soft tissue swelling and pain and may require surgical removal. US may aid in diagnosis. The molars are not assessable by US due to the buccal space.

Continuous US monitoring of the mammary gland during pregnancy may predict the time of parturition based on the morphological changes in the mammary parenchyma. A few days prior to birth large blood vessels may be detected and, within the last 24 hours before birth, large irregularly shaped cavities appear inside the glands. The color-flow Doppler mode allows differentiation between blood vessels with circulating blood and static fluid-filled cavities. Mammary gland scanning requires thorough hygienic preparation to avoid infection. Mammary gland tumors have not been found as yet by the author in Asian or African elephants.

After 12 months, the fetus sinks deep to the abdominal ventrum and becomes visible by transcutaneous US, although visualization by transrectal US is limited. Transcutaneous US provides the best image of the fetus after 16 months. The fetal position in the uterus determines the side from which US is best performed. Transcutaneous US of late pregnancy is accomplished through a specific triangular "window" defined by the caudal extent of the rib cage, ventral abdominal wall, and cranial aspect of the femoral muscle.[30] Sometimes the accessibility greatly improves if the female lifts the hindleg on the side where the examination is performed (Fig. 27.2). In the late stage of pregnancy, the uterus is positioned directly against the ventral abdominal muscle wall, which has an average thickness of 25 mm (Fig. 27.8). Besides the fetal membranes and fluid, the head and trunk, thorax with a beating heart, tail and feet may

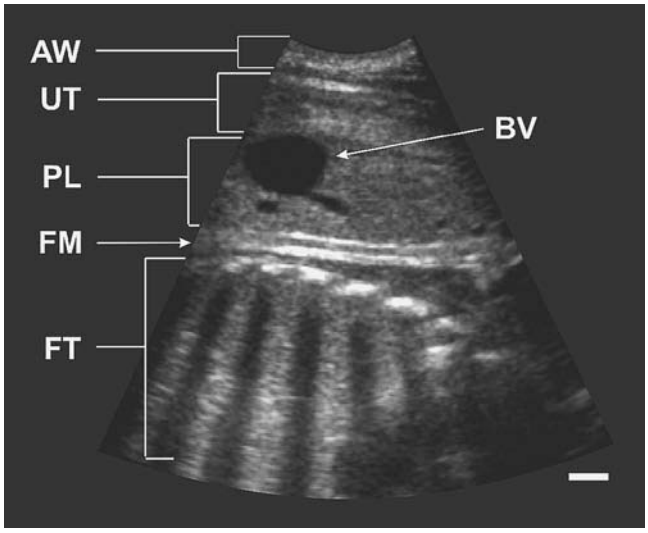


***Figure 27.8.*** *Transcutaneous sonogram (4-2 MHz) from a 16.5 months pregnant African elephant. The abdominal wall (AW), including the skin, is most near the transducer. The uterus (UT) appears as an approximately 25 mm thick strip directly below the abdominal wall (AW). The placenta (PL) contains large blood vessels (BV) and is tightly attached to the uterus (UT). Echogenic-appearing fetal membranes (FM) are located between the placenta (PL) and the fetal thorax (FT). At that fetal stage only parts of the fetus can be visualized sonographically.*

*NOTE: All white bars in the right corner of the sonograms demonstrate the distance of 20 mm.

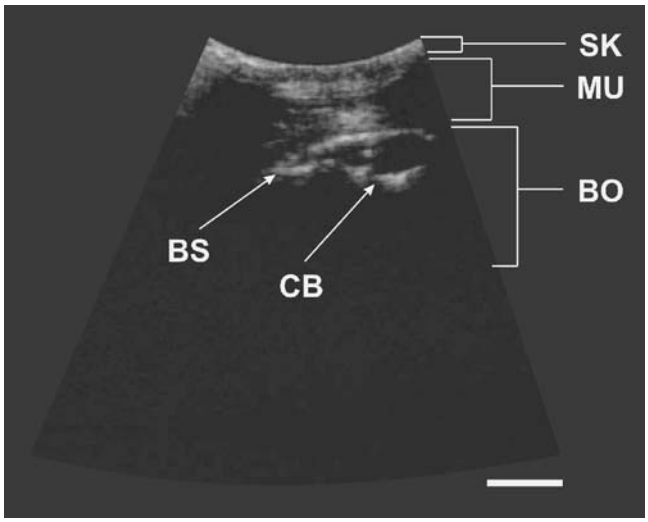


***Figure 27.9.*** *Transcutaneous sonogram (4-2 MHz) of the elbow in an 18 year-old Asian elephant bull with dramatic joint pathology. The skin (SK) and the musculature (MU) can be imaged by the US. Of the bone (BO), only the bone surface (BS) is visible. However, the appearance of the irregularly shaped bone surface (BS) in combination with the presence of a cystic bone structure (CB) indicates clearly the severe damage in the elbow joint. The male was finally euthanized.*

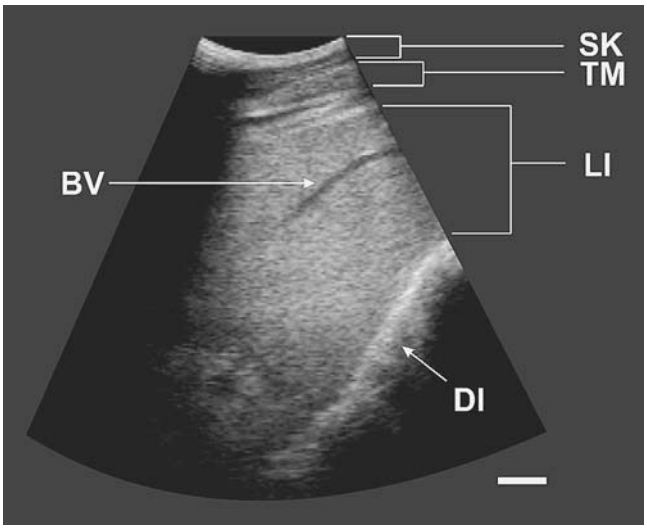


***Figure 27.10.*** *Transcutaneous sonogram (4-2 MHz) of a part of the liver (LI) in a sick Asian elephant. Through the skin (SK) and the thoracal muscles (TM), a good image of the swollen liver located next to the diaphragm (DI) was visible. The organ appears moderately echogenic indicating fatty degeneration and contains enlarged blood vessels (BV). An US-guided liver biopsy would be possible under such condition.*

be easily identified. Important for monitoring fetal health is the ultrasonographic detection of fetal movement, especially around the expected parturition date. Normally fetal movements occur about every 2 minutes and are clearly distinguishable from maternal peristalsis and muscular contractions. The status of the calf (i.e., live or dead) is of paramount importance in determining the protocol for the impending birth, including possible surgical intervention. Pregnant elephants should be conditioned for at least the transcutaneous US monitoring regardless of their management setting.

In adult elephants, the successful application of radiological joint and bone diagnostics are limited to the feet and the tusks.[5] The other regions, such as the elbow or knee joints, are too solid for conventional X-ray machines. US may be used to evaluate the integrity of joint surfaces and may detect enlarged joint spaces or fractures (Fig. 27.9).

The position of the low-frequency (4-2 MHz) scan head depends on the anatomical structure of the joint and the region of interest. All joint-associated soft tissue such as ligaments, tendons and sheaths, muscle, and fascia may be evaluated with US. Bone material has a high echodensity; thus the bone margin causes a significant impedance jump resulting in angle-dependent total reflection of US beams. Normally, only the contour of the bone is visible as a hyperechogenic line. Due to this fact, only the surface of the bones may be evaluated. Periostial lesions (i.e., exotosis, fissures) and fractures, as well as resorptive lesions involving the bone surface, may be diagnosed with US. Additionally, although the feet can be sufficiently X-rayed, transcutaneous US allows the identification and localization of prominent blood vessels in the lower appendages, which are important for the application of localized antibiotic infusion therapy for treating osteomyelitis.

Peripheral aspects of the liver and heart are accessible by transcutaneous US through the intercostal space. For this procedure the elephant must be trained to stand with one foot on an elevated platform (common elephant stand) in order to open the viewing window. This examination is relevant with suspected heart disease (i.e., pericarditis, pericardial effusion).[31] US may also aid in evaluating cardiac rhythm; however, the major internal part of the heart cannot be imaged. The detection of enlarged blood vessels, rounded liver lobe margins (Fig. 27.10), and blood congestion (increased amount of blood in the liver with reduced or even retrograde blood flow) with Doppler mode US is indicative of congested heart problems in elephants with chronic heart disease or anesthetic recovery failure.

Diseases of the liver—such as hepatic tumors, hepatic necrosis/sclerosis, parasitic liver flukes (common in Asian camp elephants) or parasitic oocytes in the liver,[31] hepatitis, and hepatomegaly—may be diagnosed using transcutaneous US when the portions of the liver imaginable by US are involved. The integrity of the capsule and the contour of the liver may be evaluated. Unpublished attempts to image the kidneys transcutaneously from the dorsum of the elephant proved unsuccessful because of their retroperitoneal location (personal communication, C. Thitaram). However, the kidneys are easily imaged with transrectal US.

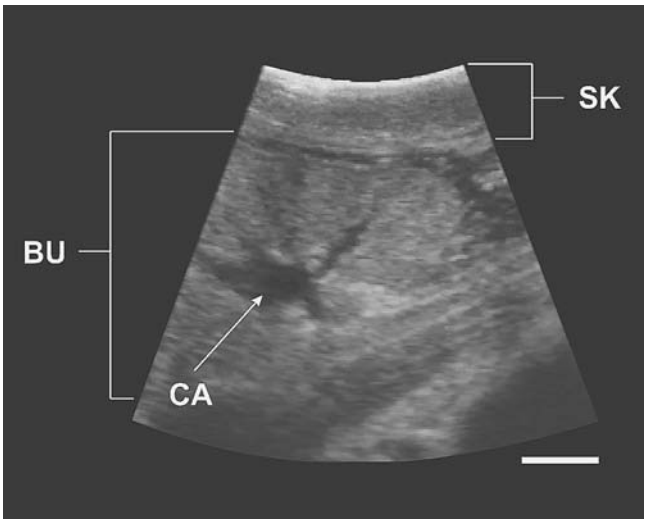


***Figure 27.11.*** *Transcutaneous sonogram (3.5 MHz) of a part of the bulbourethral glands (BU) in an adult African elephant bull. Through the skin (SK), a good image of the gland parenchyma and the irregularly shaped cavity (CA) can be generated. The parenchyma appears moderately echogenic in contrast to the unechoic cavity containing the sticky secretion that will be released prior to the penile intromission.*

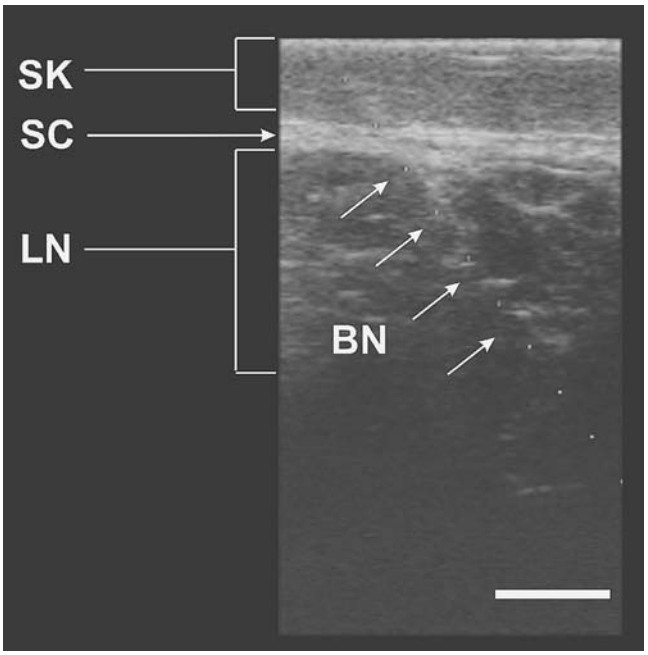


***Figure 27.12.*** *Transcutaneous sonogram (3.5 MHz) of the retropharyngeal lymph node generated with a specific biopsy transducer and the US system EUB405, Hitachi, Inc., during a fine-needle biopsy (16 gauge) procedure. The skin (SK) appears as a moderate echogenic strip followed by the echogenic subcutis (SC). In general, lymph node (LN) tissue appears less echoic. The inserted biopsy needle (BN) is causing a strong echo marked with the white arrows inside the lymph node parenchyma.*

The only assessable parts of the urogenital tract for transcutaneous US are the following: in male elephants, the paired bulbourethral glands located directly below the anus on both side of the urethra, the penile urethra, and the penis; in females, the vestibule (urogenital canal) and the clitoris with glans clitoridis. The size and the developmental stage of the bulbourethral glands is a good indicator for the current sexual status of the bull. Large glands with a well-developed, irregularly shaped internal cavity are characteristic of breeding bulls. The bulbourethral gland parenchyma has moderate echogenicity. Many secretory channels empty into an irregular medullary cavity (Fig. 27.11). The lumen of these channels and the cavity contain thick, clear secretion, but it appears surprisingly anechogenic. The visualization of the urethra in the perineal region may be important to diagnose urinary caliculi blocking the urethral lumen. The diagnosis of vestibular polyps in females is possible by transcutaneous US; however, vestibular endoscopy is the gold standard.

Transcutaneous US may be used for guiding aspiration or biopsy instruments. This technique is frequently used for diagnostic purposes for collecting lymph node tissue for the endotheliotropic elephant herpes virus (EEHV)[25] PCR-test in nondiseased elephants.[17] The technique includes the identification and morphometric measurements of the superficial retropharyngeal lymph node as well as the precise biopsy of a selected part of the lymph node parenchyma by means of a biopsy device and the needle guidance option of the US system used (Fig. 27.12).

## TRANSRECTAL ULTRASOUND IN ELEPHANTS

### Urethra

The urethra in both female and male elephants is a well-defined structure with a strong internal sphincter. In females, the total length is 80–110 mm and the diameter is 30–40 mm (Fig. 27.13). In males, the total length is on average 2.0 m with a fully erected penis.

The diameter of the pelvic urethra in males is 35–55 mm. In the cross-sectional sonogram, the urethra is recognized as a round structure, surrounded by a distinct layer (2–8 mm) of hypoechogenic tissue (muscle). Most of the urethra appears hyperechogenic, except for an irregular, V-shaped hypoechogenic center (mucosal folds), which is about 5 mm in diameter. In addition, there is a seminal colliculus in elephant bulls detectable only in cross-section as a hypoechogenic area on the dorsal wall of the urethra about 20–50 mm caudal from the neck of the urinary bladder. It measures approximately 27 mm wide and 15 mm deep. The organization of the urethra appears identical in longitudinal sonograms. The longitudinal view of the urethra in both sexes is important for the evaluation of the mucosal integrity (urothel).

About 10% of the assessed animals have shown inflammatory processes or chronic calcifications inside

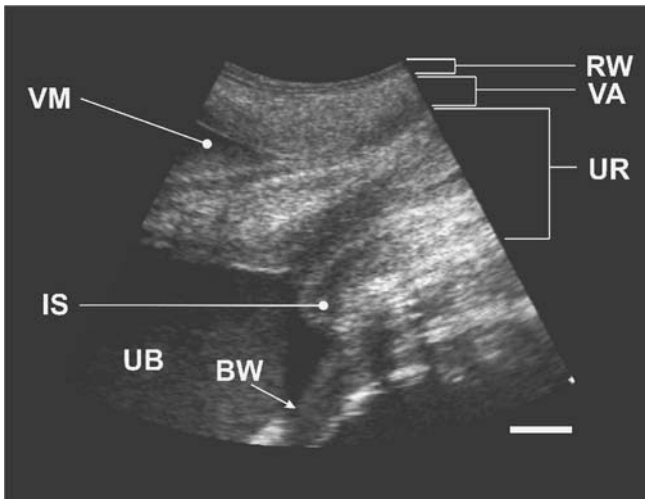


***Figure 27.13.*** *Transrectal sonogram (4-2 MHz) of the pelvic region shows the urethra (UR) with the internal sphincter (IS), urinary bladder (UB) with its bladder wall (BW), and the beginning of the cone-shaped vagina (VA). The sonogram was taken at the end of the luteal phase visible by the presence of the vaginal mucus (VM). The rectal wall (RW) appears as a moderate echoic strip on top of the sonogram.*

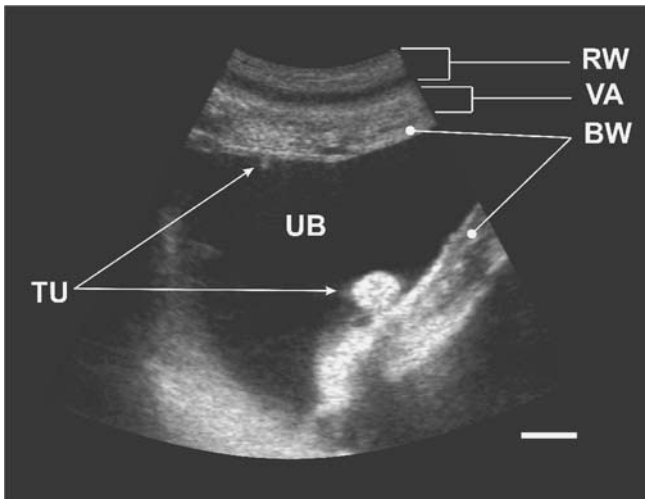


***Figure 27.14.*** *Transrectal sonogram (4-2 MHz) of the urinary bladder (UB) with its bladder wall (BW) containing several tumors (TU) caused by a severe pseudomonas infection. The rectal wall (RW) appears as a moderate echoic strip on top of the sonogram.*

the pelvic urethra. These alterations have no direct impact on the capability of the cow to conceive or the bull to mate; however, they may become extensive and painful interfering with breeding behavior. In newborn elephants, the urethra measures 4 mm in diameter and the lumen appears uniformly rounded in cross-section, rather than V-shaped as in the adults.

## Urinary Bladder

The urinary bladder is pear-shaped (150–450 mm long, 100–300 mm wide, and 100–300 mm deep) and has a relatively small volume capacity in comparison to the body size. This explains why elephants urinate frequently. The bladder is anchored by ligamenta and connective tissue and does not prolapse in contrast to the genital structures.[20] Sonographically, urine appears mostly anechogenic and sometimes slightly cloudy depending on the diet. The hyperechogenic bladder wall is about 3–10 mm thick depending on the degree of expansion. The differentiation of the three smooth muscle layers is evident using a high-frequency transducer (7.5 MHz or higher) only in the captive males. Urinary bladder infections or tumors are relatively rare and affect mainly older elephants, but they are easy to diagnose (Fig. 27.14). US follow-up examinations allow monitoring of the healing process during treatment.

The caudal parts of the ureters are integrated into the wall of the bladder and are readily distinguishable. They are 4–6 mm thick and appear less echogenic than the dorsal wall of the urinary bladder. In individuals that are nervous during the US examination, there is frequent transport of urine from the kidney to the bladder. Pathological alterations of the ureters have not yet been diagnosed sonographically in elephants; however, it is prudent to check them with every US examination.

## Kidneys

The dorsal sides of the lobulated kidneys are retroperitoneal, close to the rectum, and approximately 1.5–2.0 m cranial to the anus. The kidney can be identified by its smoothly curved hyperechogenic capsule, 1–2 mm thick. The parenchyma contains several channels and blood vessels. Shadows created by the high echogenicity of the septa, which divide the renal lobes, interrupt the ultrasonographic image of the kidney. In general, imaging of the kidney is limited using a 7.5 MHz probe, and sonograms of a single kidney lobe may be easily misinterpreted as a gonadal structure, such as a large corpus luteum or testicular parenchyma. To avoid misinterpretation, the kidneys should be scanned with a low-frequency probe (3.5 MHz). In all adult elephants, parts of the kidneys may be visualized only by using the 450 mm probe extension. In adolescent elephants, the kidneys may be visualized with low-frequency and middle-frequency transducers without an extension because of their short distance (850 mm, maximum arm length) from the anus. Pathological alterations on the kidney, such as degenerative processes or tumors, may be identified ultrasonographically (Fig. 27.15).

## Male Reproductive Tract (See Figure 27.16)

**Prostate gland.** The prostate is a three-lobed gland bilateral to the urethra connected by an isthmus prostatae and located above the pelvic urethra, caudal to the ampullae and approximately 80 mm from the urinary bladder. The only notable anatomical difference between the urogenital tract of African and Asian male elephants is the size

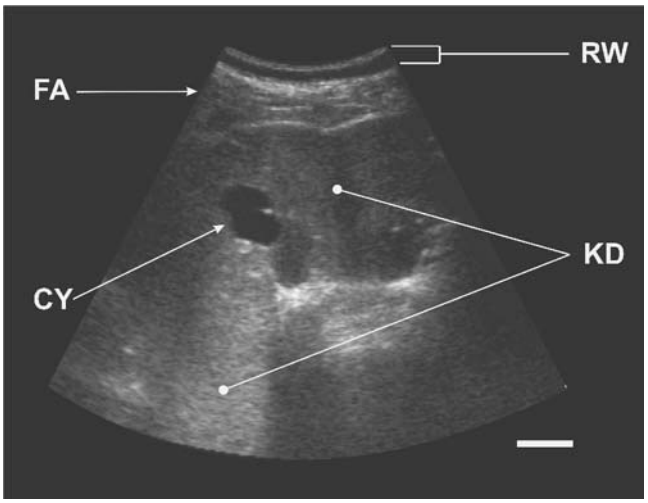


***Figure 27.15.*** *Transrectal sonogram (4-2 MHz) of a cystic degenerated kidney (KD) in an approximately 60 year-old female elephant. The cysts (CY) are clearly visible as anechoic, well-boarded, round structures inside the renal parenchyma. The kidney (KD) is surrounded by few amounts of renal fat (EA). The rectal wall (RW) appears as a moderate echoic strip on top of the sonogram.*

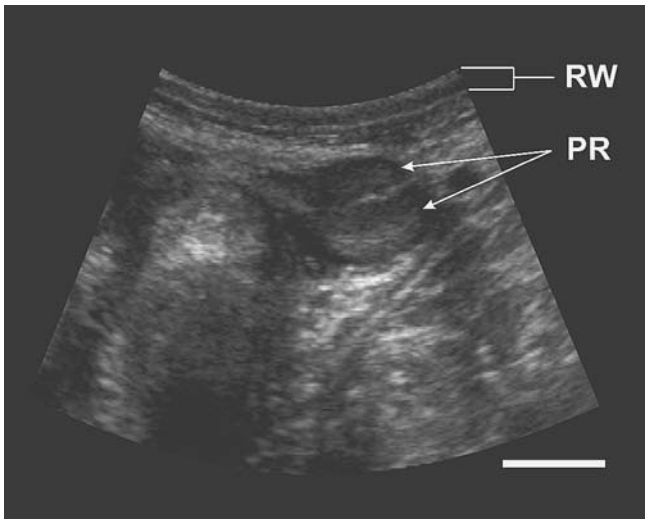


***Figure 27.17.*** *Transrectal sonogram (4-2 MHz) of solid prostate (PR) lobes in an Asian elephant bull. The prostate is much smaller than in the African counterpart and does not contain any kind of cavity. The rectal wall (RW) appears as a moderate echoic strip on top of the sonogram.*

and shape of the prostate gland. In the Asian elephants the three lobes on either side of the urethra are fused into a globe-shaped solid structure with a maximum diameter of 20 mm (Fig. 27.17). By contrast, the prostate in sexually mature African elephants has clearly distinguishable lobes with separate, irregularly shaped internal cavities (Fig. 27.18). These paired structures located on either side of the urethra may be as large as 60 mm in diameter.

The entire prostate is visible only with the 4–2 MHz probe, but the 7.5 or 10.0 MHz probe is required to detect its detailed structure. The prostate consists of a thin, hyperechogenic capsule of connective tissue surrounding a 1–2 mm hypoechogenic muscle layer, which is attached directly to the stroma. In African elephants, the moderately echogenic, highly folded stroma of each lobe surrounds a large, irregularly shaped, fluid-filled

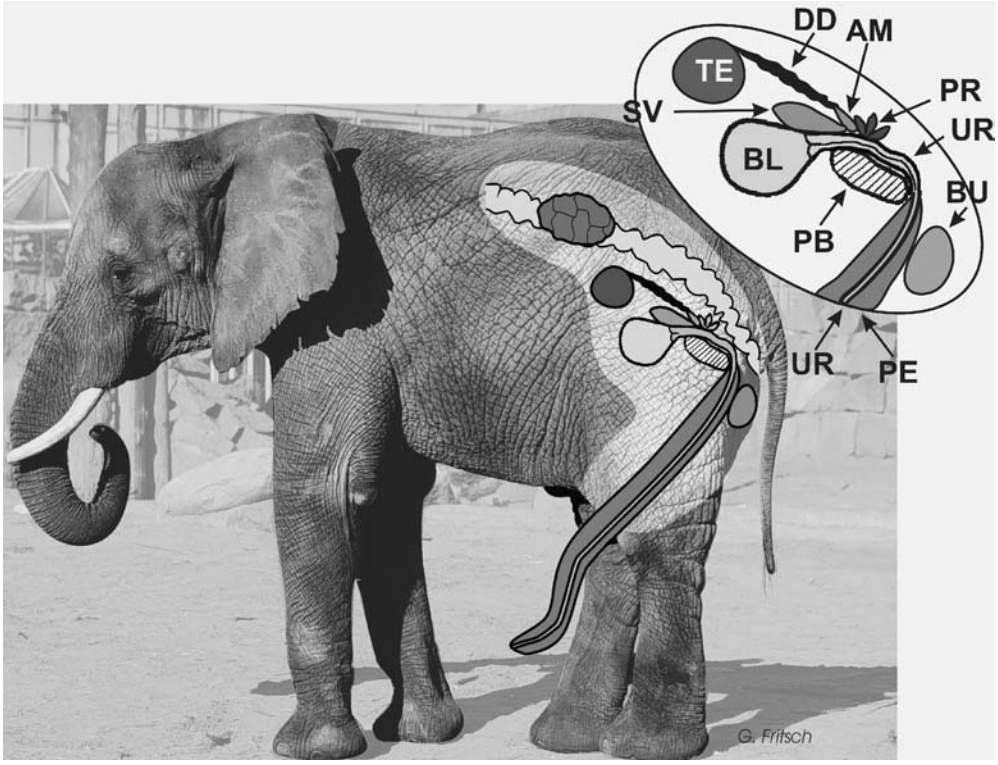


***Figure 27.16.*** *Schematic diagram of the different organs of urogenital system in male elephants: testis (TE), ductus deferens (DD), ampulla of the ductus deferens (AM), seminal vesicles (SV), lobulated prostate (PR), urethra (UR), bulbourethral gland (BU), penis (PE), pelvic bone (PB), and urinary bladder (BL).*

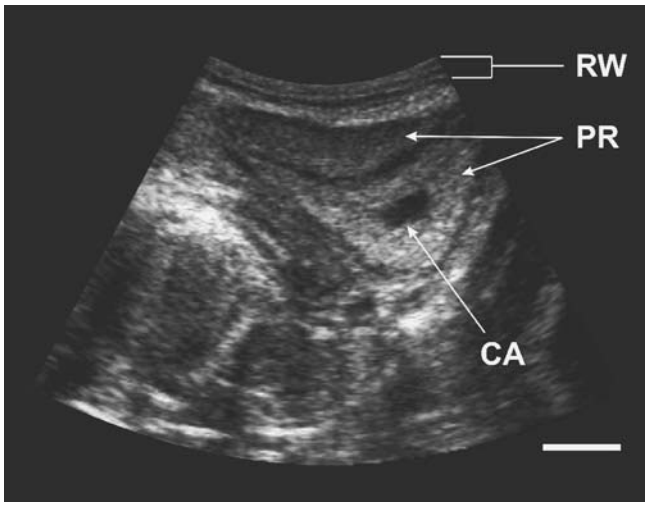


***Figure 27.18.*** *Transrectal sonogram (4-2 MHz) of the lobulated prostate (PR) of an African elephant bull. Note the irregularly shaped internal cavity (CA) in each prostate lobe in comparison with the solid prostate in Asian elephants. The rectal wall (RW) appears as a moderate echoic strip on top of the sonogram.*

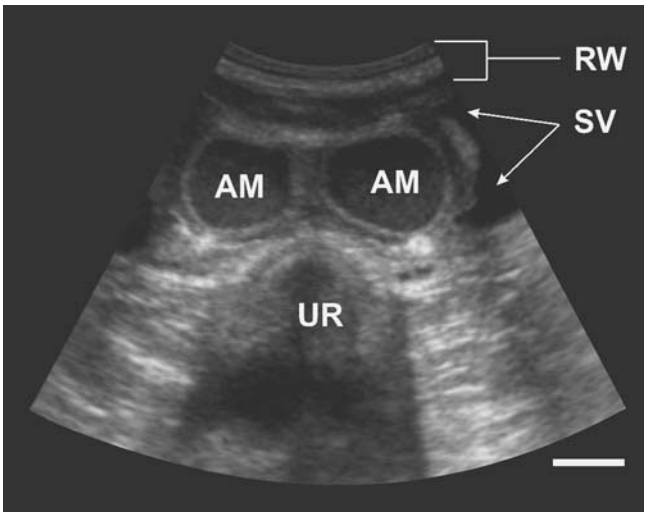


***Figure 27.19.*** *Transrectal sonogram (4-2 MHz) of the ampullae (AM) region taken in cross section. If the ampullae are filled, the image appears as a schematic "Mickey Mouse" (hidden Mickey Mouse image) in combination with the circular-shaped urethra (UR). The seminal vesicles (SV) are in the cranio-lateral position. The rectal wall (RW) appears as a moderate echoic strip on top of the sonogram.*

cavity, which appears anechogenic. There is a dramatic developmental difference in size and complexity of the glands between adolescent and adult males. Together with the seminal vesicles, the prostate is the last to develop during puberty, and it develops only after the testicular diameter reaches 70–100 mm. The undeveloped prostate gland is difficult to identify from the surrounding connective tissue of the urethra. For clear imaging of the gland at this stage, high-resolution transducers (10.0 MHz) are required.

**Ampullae.** In elephants, the cone-shaped ampullae (terminus of the ductus deferentes) store spermatozoa produced in the testes and matured in the coiled ductus deferens system.[19] The ampullae are located bilaterally dorsal to the caudal part of the urinary bladder immediately cranial to the bladder neck. If both ampullae are fully filled and the bladder is expanded, these structures create a characteristic “Mickey Mouse” image using a 4–2 MHz probe (Fig. 27.19).

Each ampulla may reach a maximal diameter of 50 mm and a maximal length of 70 mm. The appearance and dimension of the ampullae is a good indicator of the current reproductive status of the bull being examined. If the glands are fully dilated and have an organized snowy echogenic appearance, this indicates the presence of high sperm concentration. This echo reflectivity is typically seen in bulls with active testes. In the absence of spermatozoa, the lumen of the ampullae has an anechogenic appearance, characteristic for prepubescent animals in which the glands produce a sperm-free secretion. Under the influence of testicular development, the ampullae become active when the testicles are approximately 50 mm in diameter; therefore, before this stage no fluid is present in the lumen and the ampullae are undistinguishable from the surrounding connective tissue.

The nature of the ampullae may be used as predictors of the amount of spermatozoa obtainable through manual collection, thus making these glands one of the most important structures to assess testicular activity indirectly. If testicular function in a mature bull is disturbed due to disease or antiaggression treatment, the morphology of the ampullae may also be affected bilaterally. Interestingly, even castrated adult males with atrophic sexual glands have sperm-free fluid-filled ampullae. In addition to testicular-insufficiency–induced disorders, unilateral abnormalities in the ampullae sporadically found are thought to be associated with disturbance during formation and/or development of the reproductive tract. A definitive congenital alteration of the ampullar region has been described. A 10 mm diameter, pea-shaped, fluid-filled structure with a clear border between the two medial ampullae walls that did not empty or change shape after ejaculation as in the other bulls, nor did it affect semen quality, was diagnosed potentially as vestigial embryonic uterine tissue (i.e., Muellerian duct, *utriculus masculinus*).

**Seminal vesicles.** The paired cigar-shaped seminal vesicle glands (Fig. 27.20, see also Fig. 27.19) are normally the largest accessory glands of an active breeding bull and may contain up to 400 ml fluid in each gland.

The seminal vesicles are the most cranially located accessory sex glands, dorsolateral to the urinary bladder. These glands are characterized by a single cavity with a wall 5–10 mm thick. An outer muscular and an

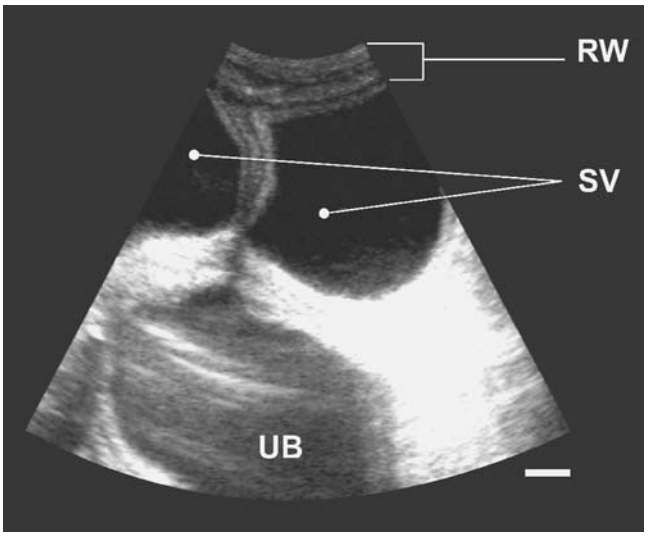


***Figure 27.20.*** *Transrectal sonogram (4-2 MHz) from the seminal vesicles (SV) in cross section located dorsal to the urinary bladder (UB). These glands are the largest accessory sex glands in elephants and filled with secretion in breeding bulls. The gland wall is well developed and shows two distinguishable layers. The hypoechoic external muscle layer and the echoic internal mucosa. The rectal wall (RW) appears as a moderate echoic strip on top of the sonogram.*

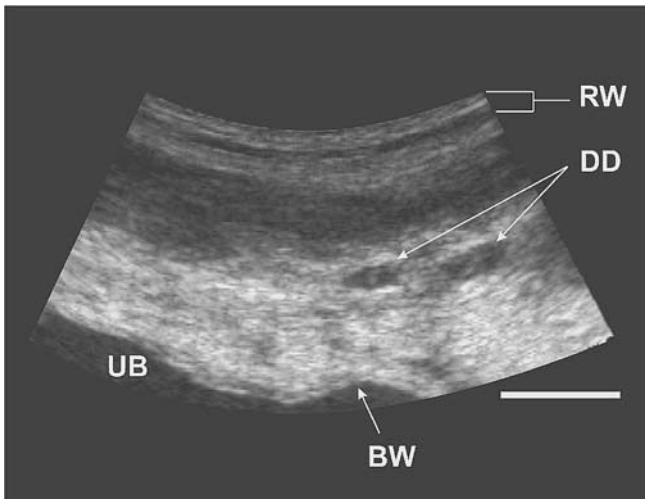


***Figure 27.21.*** *Transrectal sonogram (4-2 MHz) from the ductus deferens (DD) in cross section located dorsal to the urinary bladder (UB) with its bladder wall (BW). The internal mucosa of the ductus deferens appears as the echogenic center in comparison to the less-echoic muscle layer. The rectal wall (RW) appears as a moderate echoic strip on top of the sonogram.*

internal mucosal layer may be clearly distinguished by US. The seminal vesicles produce the bulk of the seminal fluid, which provides nutrients and a vehicle for the ejaculated sperm. These glands show the most variation of any structures of the male reproductive tract among adult individuals and are important criteria, in combination with the morphology of the ampullae, for characterizing the current reproductive status of the bull.

The maximum diameter of the seminal vesicles is about 100 mm, but the pressure of the probe on the rectal wall may slightly compress the glands. The length is difficult to measure ultrasonographically, but may extend for up to 450 mm. The vesicular wall is characterized by a 5–6 mm thick hypoechogenic muscle layer and a 3 mm thick hyperechogenic mucous membrane.

The fluid appears less echogenic than the sperm-rich fluid in the ampullae; however, in fully developed breeding bulls the seminal vesicles' secretion may appear slightly echogenic. Seminal vesicles are not filled in adolescent elephants or reproductively inactive bulls, often indicating temporary infertility.

In a healthy bull, the seminal vesicles combined empty about 50–200 ml of fluid per ejaculation after natural breeding or manual collection.[27] Even after multiple ejaculations, fluid in the lumen of the seminal vesicles may still be detected with US. Electroejaculation under anesthesia may completely empty the seminal vesicles, producing ejaculates approaching 800 ml, although ejaculate volumes in the range of 100–200 ml are more common.

**Ductus deferentes.** The ductus deferentes are positioned ventromedial to the seminal vesicles and dorsal to the urinary bladder (Fig. 27.21) and empty into the ampullae. They are surrounded by connective tissue and large fat pads.

Ultrasonographic cross-section using 7.5 MHz transducer is the best method to visualize these tubular structures due to the tight coiling, which makes longitudinal imaging difficult. Immediately before the ampulla–duct deferens junction, the posterior end of each ductus deferens is 5–10 mm in diameter in the adult males. Three regions are distinguishable: the anechogenic lumen (if visible, it indicates testicular sperm production); hyperechogenic, irregular mucosa of the duct; and the hypoechogenic, 2 mm thick muscle layer. The middle portion of the ductus deferens is outside the field visible by transrectal US. The anterior part of the ductus deferens, near the epididymis, can be visualized only with the use of the high-frequency probe in combination with the extension in the adults. This part is 5–8 mm in diameter and is generally hypoechogenic without clear distinction between epithelium and muscle layers. In adolescent males, the ductus deferens is similar in organizational structure to those of the adults except that the lumen is not filled with spermatozoa and the reduced dimension (<5 mm diameter) may be visualized with handheld high-resolution transducers. However, the ductus deferens is not as an effective indicator of breeding potential as the ampullae and the seminal vesicles.

**Epididymides.** Ultrasonographic imaging of the epididymis is possible only with the 450 mm extension with the 7.5 MHz linear probe. The elephant epididymis

is not prominent compared to the species. Additionally, the epididymis is not well demarcated from the rest of the genital duct in the elephant, making it difficult to locate.[18,19,28,29] The epididymis is approximately 100 mm long and 10–20 mm wide, surrounded by a large amount of hyperechoic connective tissue. The epididymis is characteristically hypoechogenic. The best method for locating the epididymis is by scanning the entire testicle. Sonographically, there is no apparent differentiation between the caput, corpus, and cauda epididymidis.

**Testes.** Elephants have internal testes situated caudoventral to the kidney. They are spherical and may reach a maximum diameter of about 150–230 mm.[11,13] Testicular tissue is visualized using low-frequency transducers for overview and high-frequency transducers for detailed examination of the parenchyma. In adult bulls, the 450 mm adapter is necessary (Fig. 27.5) due to the anatomical location of the intraabdominal testes approximately 0.9–1.5 m from the anus. The hypoechogenic testicular parenchyma appears homogenous, divided by fanlike projections of moderately echogenic septa, and is bordered by the highly echogenic, slightly convex tunica albuginea. The mediastinum testis (corpus fibrosum) is an irregular hyperechogenic region at the core of the adult testis, approximately 30 mm long and 10 mm wide with moderately echogenic septa branching toward the periphery (Fig. 27.22). The testes of adolescent males (Fig. 27.22) may be visualized without the use of the probe extension and middle-frequency probes (7.5 MHz) due to the shorter distance from the anus and the small diameter of the testes. The internal structure of the subadult testis shows remarkable differences compared to adult gonads (Fig. 27.23). In the subadult, a central major blood vessel (2 mm in diameter), embedded in the rete testis, is detectable with the 3.5 MHz probe. This central blood vessel is not detectable by US or in the gross dissection of the testes in adults.

Testicular size and the appearance of the parenchyma is usually a good indicator of reproductive status; however, in many bulls it is not possible to visualize the gonads due to interference from ingesta-filled intestinal loops or caecum. An alternative option to visualize both testes in bulls in which direct contact is possible or in fully anesthetized bulls is to perform the examination in lateral recumbency using also the 450 mm probe extension and low-frequency probe (4-2 MHz). There is a slight difference in dimension between right and left testes. The right testis has a globe shape in contrast to the slightly egg-shaped left testis. However, there was a case described by Hildebrandt and colleagues[13] of remarkable difference in testicular diameter between the right (160 mm) and the left (100 mm) testes. Further, this difference caused a difference in the degree of filling in the corresponding right and left ampullae. Interestingly, this Thai camp bull was otherwise physically mature and a proven breeder; the cause of this disproportion was probably a developmental disorder.

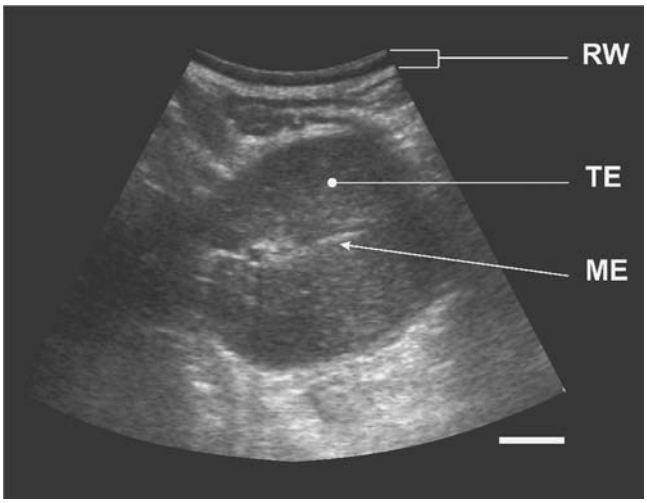


***Figure 27.22.*** *Transrectal sonogram (4-2 MHz) of the right testis (TE) in an adolescent male elephant. The juvenile testis has a much closer proportion between the mediastinum testis (ME) and the testicular parenchyma than in an adult testis (Fig. 27.21), indicating the absence of the spermatogenesis. The rectal wall (RW) appears as a moderate echoic strip on top of the sonogram.*

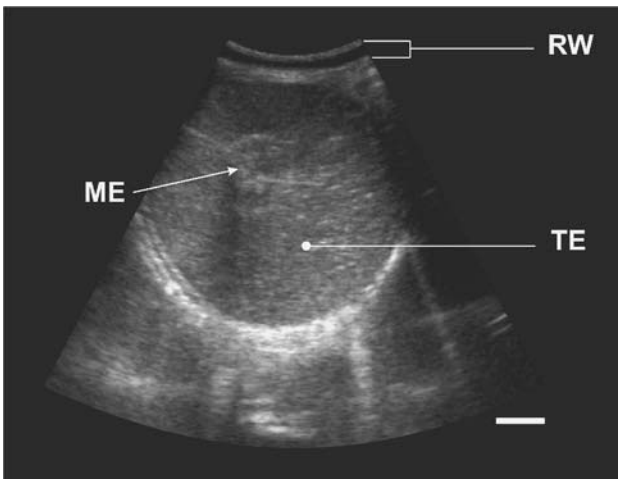


***Figure 27.23.*** *Transrectal sonogram (4-2 MHz) of an active right testis (TE) in a breeding bull. The testicular parenchyma is much more dominant in comparison to the mediastinum testis (ME). The rectal wall (RW) appears as a moderate echoic strip on top of the sonogram.*

The immature testis ranges from 20 mm (newborn) to ~90 mm (adolescents). Adequate sperm production for fertility occurs around a testicular diameter of 100 mm. In addition to the difference in size between immature and mature gonads, there is a remarkable difference in the echogenicity of the parenchyma, due to the spermatogenic inactivity and activity of the parenchyma, respectively. This phenomenon is also observed in sexually mature adult males under social suppression by

other bulls, older cows, dominant elephant handlers, or long-term antiaggression treatment. The parenchyma of an active testis appears less echoic than an inactive testis due to the higher degree of blood circulation. However, concentrations of testosterone measured in peripheral blood often do not reflect the differences in size and echogenicity between small inactive and large active testes, suggesting this measurement alone may not be useful in determining the reproductive status of a bull elephant.

Figure 27.24 (see Color Section) shows a color-flow Doppler image of the internal blood circulation of the testicular parenchyma (inside the white frame), which illustrates the high tissue metabolism in a sexually active bull. However, the results from the color-flow Doppler examinations were not markedly different from those using B-mode US.

Testicular pathology is rare in elephants. Besides developmental differences in size between left and right testes, only testicular cysts have been described (Fig. 27.25).

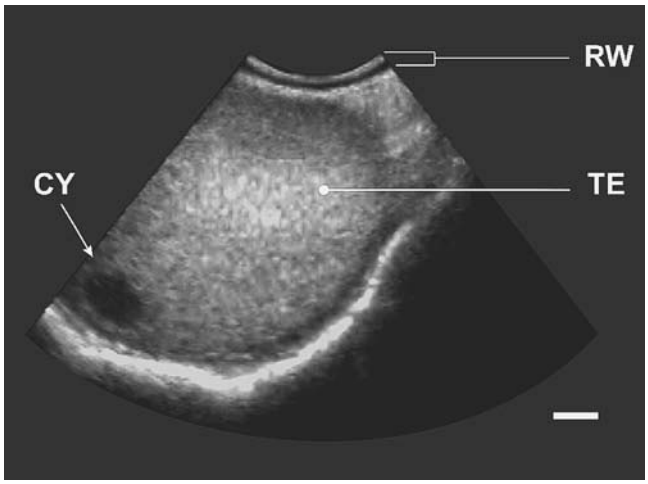


***Figure 27.25.*** *Transrectal sonogram (3.5 MHz) of a left testis (TE) containing a cystic formation (CY). This male was not a breeding male. The rectal wall (RW) appears as a moderate echoic strip on top of the sonogram.*

## Female Reproductive Tract (See Figure 27.26)

**Vestibule.** The vestibule or urogenital canal is extremely long in elephants (1.0–1.4 m). The vestibule is a tubelike structure that begins between the hindlegs, runs vertically up toward the tail, and then curves horizontally at the cranial end, creating a sac (200–400 mm) situated above the bony pelvis. The vertical part of the vestibule extending to the pelvic bone (approximately 0.8–1.0 m in length) can be visualized only transcutaneously. The clitoris is integrated in the cranial muscular wall of the vertical part and reaches a length of 0.6–0.8 m. The glans clitoris measures 70–120 mm and is situated close to the external opening of the vestibule. The horizontal part of the vestibule (approximately 200–400 mm in length) containing the urethral and vaginal orifice can be imaged only with transrectal US.

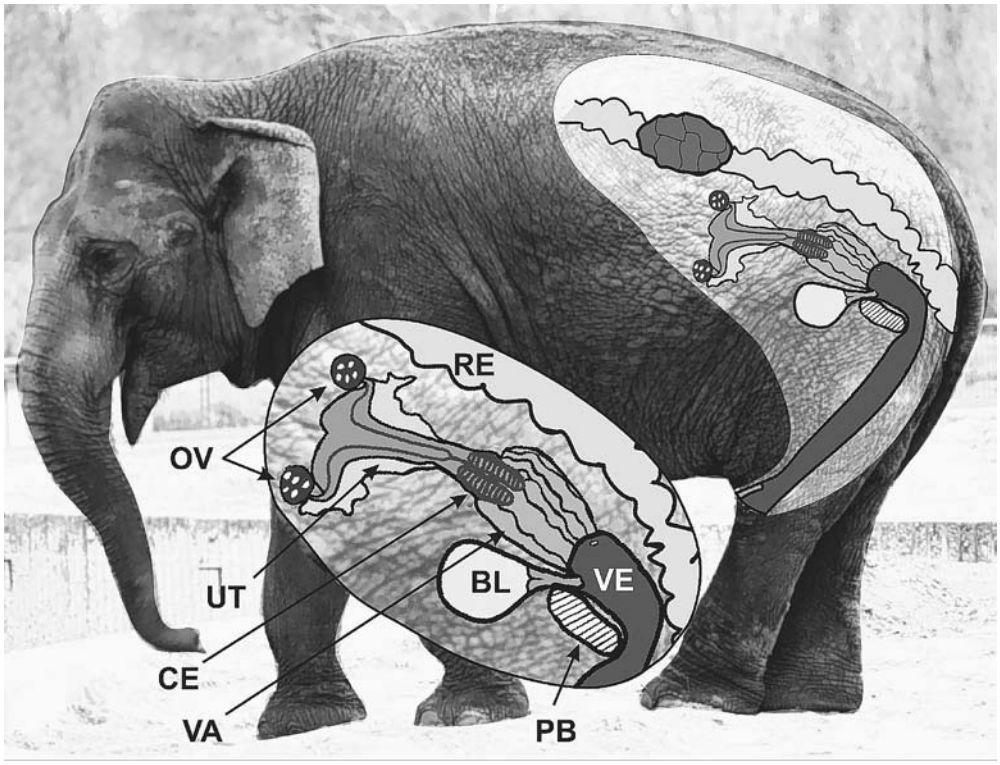


***Figure 27.26.*** *Schematic diagram of the different organs of urogenital system in female elephants: ovaries (OV), uterus (UT), cervix (CE), vagina (VA), vestibule (VE) or canalis urogenitalis, pelvic bone (PB), urinary bladder (BL), and rectum (RE).*

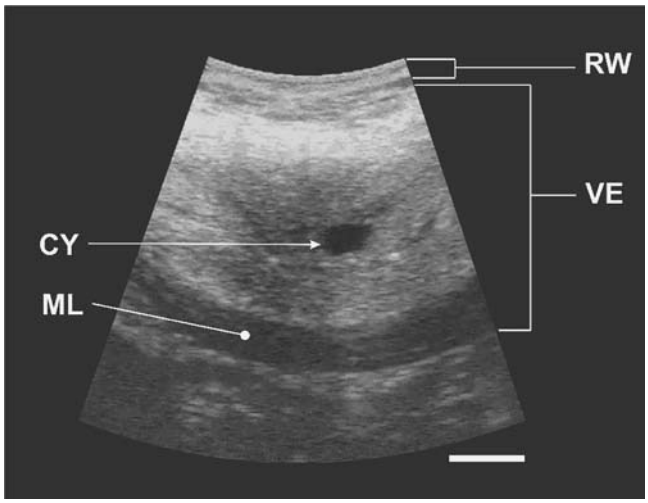


***Figure 27.27.*** *Transrectal sonogram (3.5 MHz) of the horizontal part of the vestibule (VE) in cross section (RW = rectal wall). Note the dominant outer muscle layer (ML) of the vestibule (VE) and the cyst (CY) in the vestibular mucosa. This pathological alteration is relatively common in older nulliparous female elephants.*

The horizontal portion of the vestibule is best imaged in cross-section using a low-frequency probe (4-2 MHz). The vestibule appears ellipsoid shaped in cross-section. The hypoechogenic mucosa (1 mm thick) is surrounded by a prominent hyperechogenic submucosa containing several blood vessels (5–10 mm thick), which is framed by a large hypoechogenic muscle layer reaching an approximate thickness of 10 mm (Fig. 27.27). The vestibular lumen is normally not detectable during US examination.

Numerous vestibular pathologies have been described in African and Asian elephants.[14] In general, older nulliparous female elephants have a higher incidence of reproductive pathologies than their pluriparous counterparts.[8,9,14,15,24] This includes vestibular lesions. Vestibular cysts may range from a singular mucosal cyst (Fig. 27.27) to multiple cyst formations located around the vaginal orifice and urethra. These vestibular cysts are found frequently in captive elephants of both species. Vestibular adenomas have been rarely diagnosed in both species. In contrast to these lesions, vestibular polyps, up to 50 mm in diameter, have been diagnosed only in older captive African cows (in about 70% of African elephant females >30 years of age). Vestibular scars may often be found in the pelvic rim region, the narrowest part of the vestibule, located directly below the anus. These alterations in the vestibular mucosa and/or muscle are thought to originate from a spectrum of possible causes, such as urogenital infection, mechanical injuries during mating or birth, episiotomy for the surgical approach of artificial insemination, or injury from the incorrect use of an elephant hook. Permanent vestibular fistulas caused by episiotomies or fetotomy for calf extraction (dystocia treatment) have been described for several females.[16,21,23,26]

In general, the vestibular lesions are thought to have no major direct impact on fertility of the affected females; however, these lesions may be very painful during mating and therefore have dramatic influence on the natural breeding behavior (so-called "crossing legs syndrome") and may potentially trap the calf after passage through the pelvis due to reduced vestibular elasticity or adhesion.

**Vagina.** The vagina is characterized by many longitudinal folds. It measures approximately 300 × 150 × 100 mm and serves as the place for natural semen deposition. In pregnant females, the vagina takes on the function of a mechanical and infectious protective barrier by filling with thick vaginal mucus. Nulliparous females have a hymenlike structure which does not rupture during mating. This vaginal os (Fig. 27.26) is only 4 × 2 mm and is flanked by two blind pouches (relics of the Wolffian ducts). During parturition, the hymen is stretched and ruptured by the intruding allantoic and/or amniotic sac permanently disrupting the blind pouches. One year after postpartum, the vaginal opening contracts to dimensions of approximately 10 × 10 mm.

There are significant morphological changes of the vaginal mucosa and the amount of mucus present in the lumen during cycling and pregnancy, which may be used as diagnostic criteria for determining the current reproductive stages. In general, cross-sectional images of the vagina and cervix are more difficult to interpret and not very useful. Longitudinal imaging using a low-frequency handheld probe (4–2 MHz) is the gold standard for vaginal diagnostic imaging. The vaginal lumen is partially filled with anechogenic mucus at the middle and end of the luteal phase. In contrast, during pregnancy the amount of mucus increases at least threefold. The characteristic of the vaginal lumen changes dramatically throughout the estrus cycle. Around estrus, the mucosa is enlarged, highly folded, and homogeneously hypoechogenic, demonstrating the most ultrasonographically active phase. During the early nonluteal phase, the vagina is very thin and homogeneously hyperechogenic with undetectable vaginal folds and lumen demonstrating the most ultrasonographically inactive phase.

The vagina of an adult female elephant that is hormonally acyclic ("flatliner" syndrome, with no detectable luteal or cycle activity) has an even more advanced dormant appearance. Temporary or permanently acyclic infertility affects 15% of Asian and 25% of African females,[4] predominately older nulliparous females. During the other phases of the cycle, the vaginal mucosa appears as heterogenic intermediates between completely active (hypoechogenic) and completely inactive (hyperechogenic).

Pathology of the vagina is limited to cystic forma-

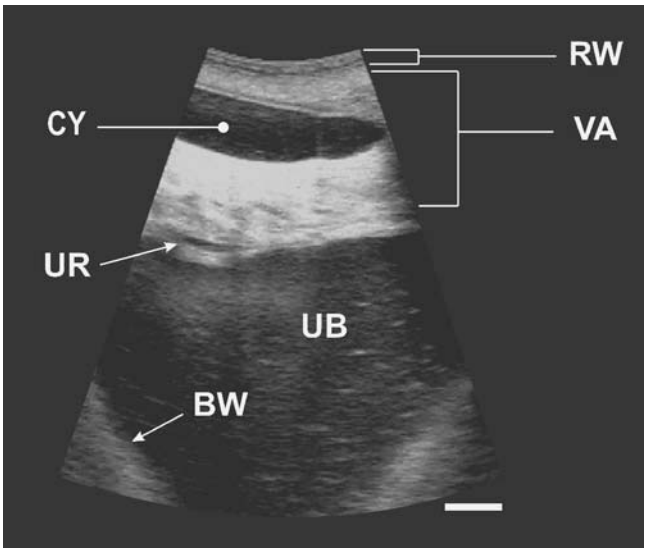


***Figure 27.28.*** *Transrectal sonogram (3.5 MHz) of the vagina (VA) and the urinary bladder (UB) with its bladder wall (BW) and the integrated ureter (UR). The vagina in this 31-year-old nulliparous Asian elephant contains two large cysts (CY), which fully fill the vaginal lumen. The rectal wall (RW) appears as a moderate echoic strip on top of the sonogram.*

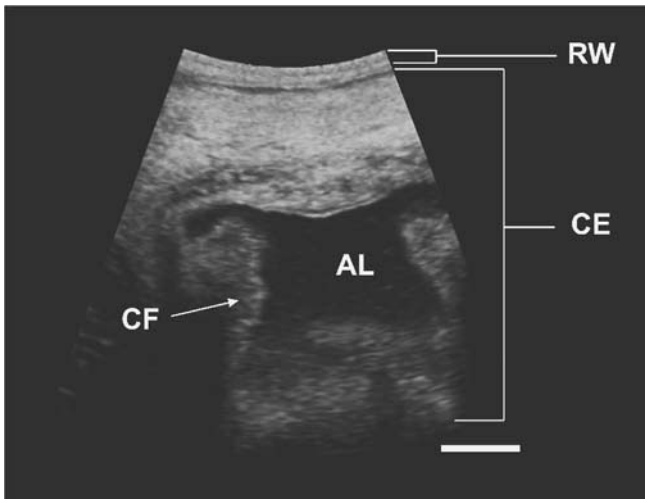


***Figure 27.29.*** *Transrectal sonogram (3.5 MHz) of the cervix (CE) in cross section during the early birthing process. The allantois sac (AL) starts to dilate the cervical canal, which allows the visualization of the cervical folds (CF). The rectal wall (RW) appears as a moderate echoic strip on top of the sonogram.*

tions and tumors. As with vestibular cysts, these lesions may be isolated small cysts (<10 mm in diameter) or extensive cystic formations (50–100 mm in dimension) filling the entire vaginal lumen (Fig. 27.28), consequently blocking the semen flow after mating and causing discomfort especially during estrus and mating. Periodic vaginal discharge containing mucus and clotted blood may be the result of such an alteration. Rarely, small mucosal tumors in the vagina can be seen during US; however, they are as relevant for infertility as the cyst formations.

**Cervix.** The cervix has a prominent portia (approximately 90 × 70 × 50 mm) but a short total length of about 150 mm. The cervix can be scanned in cross-section and/or longitudinal view using a handheld low-frequency convex scanner (4-2 MHz). Visualization of the cervical canal is important in nonpregnant females for evaluating the integrity of the cervical mucosa. The thickness of the cervical mucosa ranges from 8–15 mm and patterns the same cyclic changes as the vaginal mucosa, although it is difficult to see due to the distance of the mucosa from the scan head. Visualizing the cervical region in near-term pregnancies is especially important for determining the time of parturition. Approximately 48 hours after mechanical dilation by the amniotic sac and hormone-induced relaxation of the cervix, uncomplicated parturition should occur. Frequent monitoring of the cervix allows early dystocia diagnoses, which improves the success of the outcome of applied treatment (Fig. 27.29).

The main cervical pathology found in both species is cystic lesions. These lesions occurred more frequently in older captive African and Asian elephants (about 15% of those examined) than in wild African females (<1%). Occasionally, small cervical polyps with a maximal diameter of 10 mm are detected in African cows. One case of scarification in the cervical tissue caused by rupture during parturition was reported by Hildebrandt and colleagues.[14] Another rare birth-associated alteration in an Asian cow involving the cervix and the caudal part of the uterus was a permanent subcutaneous prolapse (large bulb under the tail) caused by partial rupture of the genital ligamenta. Female elephants trained too young to perform behaviors that result in unnatural nonphysiological abdominal contractions also tend to develop pelvic prolapses.[20] The pelvic diaphragm is not yet strong enough to accommodate the abdominal pressure, and the genital tissue becomes compressed into a bulge under the anus that can cause parturition problems later in life.

**Uterus.** The uterus is 0.8–1.5 m in length and characterized by a very short corpus uteri (50–100 mm) and two uterine horns. Both horns run parallel encapsulated in uterine serosa (false or pseudo-corpus) cranial to the corpus uteri for 0.5–0.7 m until the bifurcation. The caudal part of the uterus, including the corpus uteri and the fused uterine horns may be visualized with a handheld low-frequency transducer (4–2 MHz). The elephant probe extender (Fig. 27.5) is necessary for imaging the cranial part of the uterine horns beyond the bifurcation using low or middle frequency (<7.5 MHz) probes. The double layered mucosa of the corpus uteri (maximum 15 ± 3 mm thick) is convoluted and not as homogenous

as the endometria in the horns. Generally, the endometrium is well defined and can range in thickness from 12–45 mm. The ultrasonographic appearance of the endometrium changes dramatically throughout the sexual cycle. The influence of the hormonal cycle on the endometrial double layer was first successfully studied using transrectal US.[7,10] Under the influence of estrogens in the late follicular phase, the endometrium becomes enlarged (35.4 ± 2 mm) and appears ultrasonographically mottled with fluid presence in the cranial part of the uterine horns. During the progestin-dominated early luteal phase, the endometrium has a moderately homogenous echogenic appearance. In the late luteal phase, the endometrium begins to decrease (23.7 ± 2 mm) due to the lack of embryonic signals in a nonpregnant female. During the early follicular phase, the endometrium regresses to the smallest measured thickness (17 ± 1.5 mm) and appears hypoechogenic due to the lack of estrogens and progestins. The nonovulatory LH peak in the middle follicular phase has little to no effect on the thickness of the endometrium in contrast to the dramatic changes on the ovaries.

Pregnancies are found in the joint horn complex (Fig. 27.30) or cranially in one horn, but never in the corpus uteri. The finding that the two horns are widely separated may account for the rare phenomenon in elephants that twin. Stillborn calves may be delivered independently with a long time period between births (up to 15 months) and with both offspring not macerated by intrauterine lysis (Hildebrandt, unpublished data).

Using transrectal US, embryogenesis can be meticulously monitored. The use of the new 3D US technique further expands our ability to characterize fetal development by creating near lifelike images (Fig. 27.31). In comparison to other terrestrial mammalian species, early embryonic development appears delayed in elephants. Before 8 weeks, US detection of an embryo in an embryonic vesicle is impossible, even with US systems generating resolutions below 1 mm.

Figure 27.32 was generated approximately 1 month after birth in a wild African elephant and illustrates a

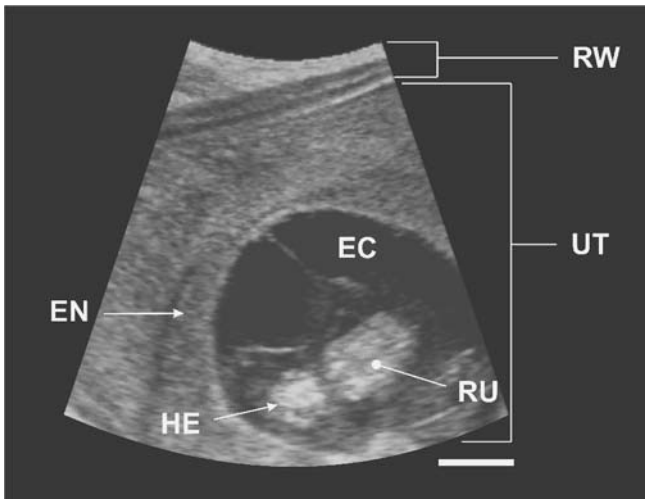


***Figure 27.30.*** *Transrectal sonogram (4-2 MHz) of the uterus (UT) containing an early embryo (approximately 3.5-month-old), which shows an already clear separation in the head (HE) and rump (RU) regions. The embryonic cavity (EC) is boarded by the endometrium (EN) and contains a lot of floating membranes forming the amnion and the allantois. The rectal wall (RW) appears as a moderate echoic strip on top of the sonogram.*

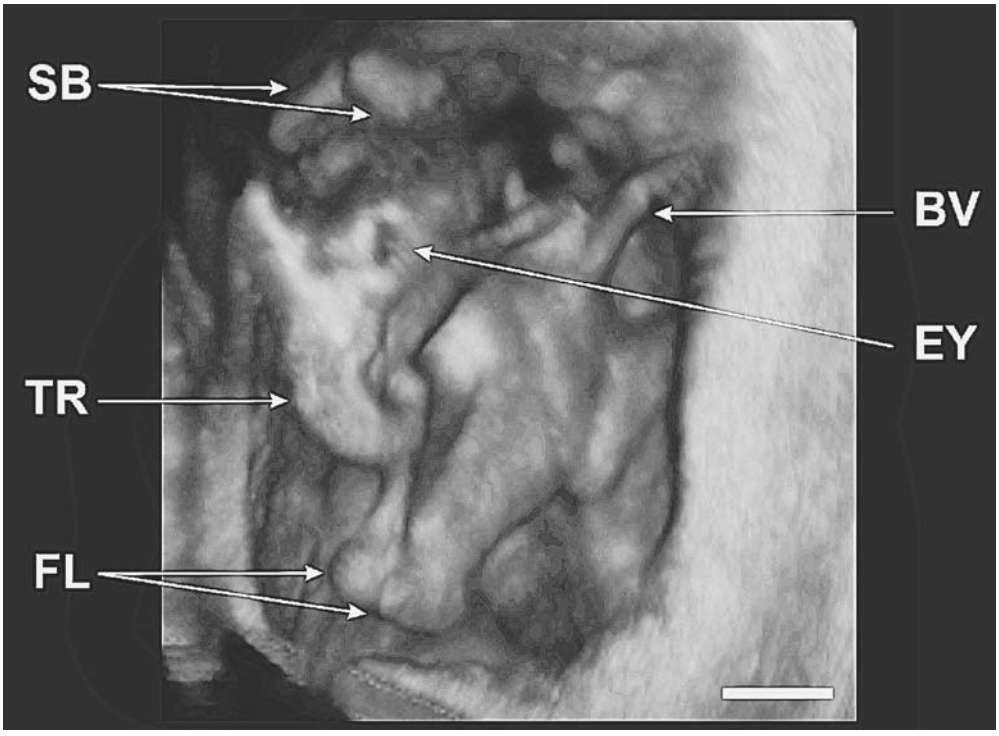


***Figure 27.31.*** *Transrectal 3D-sonogram (7-5 MHz) of a 168-day-old elephant fetus. The 3D mode allows the visualization of the fetal eye (EY), trunk (TR), front legs (FL), and skull bones (SB) as well as the external blood vessels (BV) originated from the allantois.*

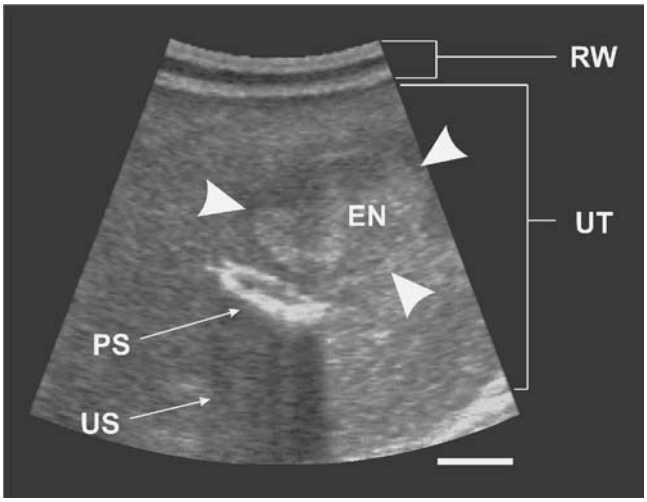


***Figure 27.32.*** *Transrectal sonogram (3.5 MHz) of the uterus (UT) approximately a month after parturition. The region of placental attachment to the uterus formats into the typical placental scar (PS), which remains a lifetime. The highly echoic scar tissue is causing an US artifact called* US shadow *(US). The endometrium (EN) appears at that stage still enlarged and relatively echogenic. The rectal wall (RW) appears as a moderate echoic strip on top of the sonogram.*

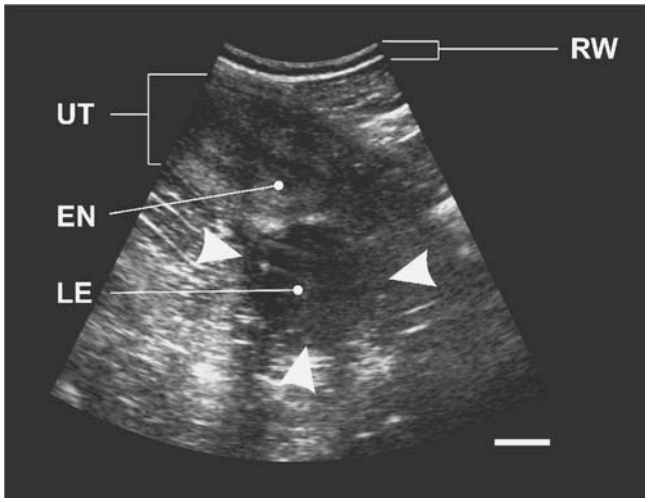


***Figure 27.33.*** *Transrectal sonogram (4-2 MHz) of the uterine horn (UT) containing a large leiomyoma (LE, marked by the white arrow heads) in the myometrium. The endometrium (EN) is not affected by this benign muscle tumor. This pathological alteration is the typical reproductive disease in older nulliparous Asian elephants as a consequence of the asymmetric aging. The rectal wall (RW) appears as a moderate echoic strip on top of the sonogram.*

uterine horn in cross-section with an enlarged endometrium and a placental scar in regression. Permanent placental scars are formed by the invasive attachment of the placenta (zonary placentation) during each pregnancy[22] and are detectable by ultrasonography or postmortem examination. Even under field conditions, the total number of placental scars may be quantitated by transrectal US, and the time of the last birth may be estimated.

Asian and African elephants exhibit clearly different uterine pathologies. Asian elephants have the tendency to develop multiple benign uterine tumors in the myometrium (*leiomyomata*) (Fig. 27.33) after long nonfertile reproductive periods (approximately 10–15 years).[8,9,14,15,24] In contrast, African elephants have not been observed with these neoformations, but rather often develop only a cystic endometrial hyperplasia (Fig. 27.34).[8,14,15] Cystic endometrial hyperplasia is also commonly found in Asian elephants.[2] A less common finding, sometimes observed in captivity but rarely in the wild (~2%), is fetal resorption or early embryonic death that resulted in temporary sterile pyometra (a noninfective, pus-filled cavity) in the affected uterine horn.[14]

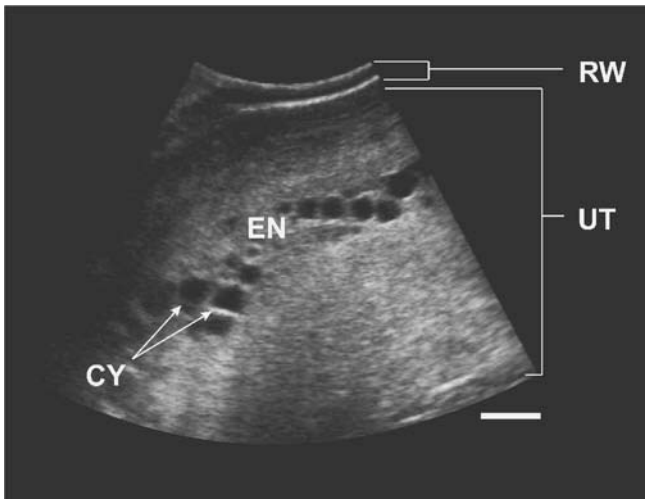


***Figure 27.34.*** *Transrectal sonogram (4-2 MHz) of the uterus (UT) with a cystic degenerated endometrium (EN). Such an endometrium can contain up to several hundred cysts (CY) and appears in older nulliparous African and Asian elephants. These individuals are permanently infertile. However, sometimes there is a local accumulation of cysts next to a placental scar, which is not causing any infertility. The rectal wall (RW) appears as a moderate echoic strip on top of the sonogram.*

**Oviducts.** The oviducts are approximately 100 mm in length. The mucosa appears less echoic than the surrounding tissue. In general, the oviducts are important landmarks for locating the ovaries in the abdominal cavity. They can be imaged only with a high-frequency transducer (7-5 MHz) in combination with the elephant probe extension. So far the only pathological alterations reported[14] are paraovarian cysts situated in the oviductal ligament. These cysts may reach a diameter of up to 50 mm in contrast to ovarian cysts, which range normally between 15 to 25 mm. Paraovarian cysts have no effect on the reproductive cycle, do not change their size in a short time period, and often occur at the end of

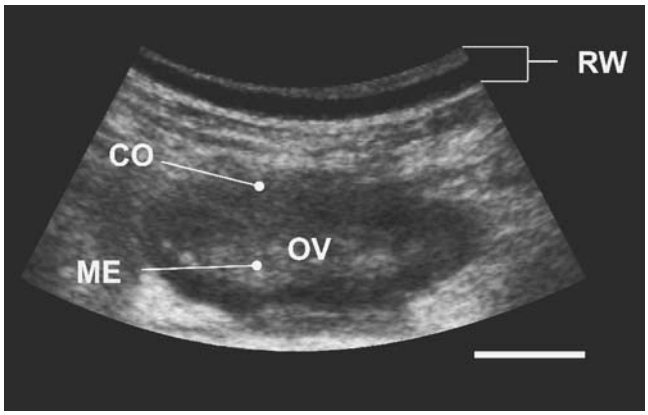


***Figure 27.35.*** *Transrectal sonogram (4-2 MHz) of a juvenile ovary (OV). It is characterized by a clear separation in the cortex (CO) and medulla (ME) and does not contain any mayor functional structures. The rectal wall (RW) appears as a moderate echoic strip on top of the sonogram.*

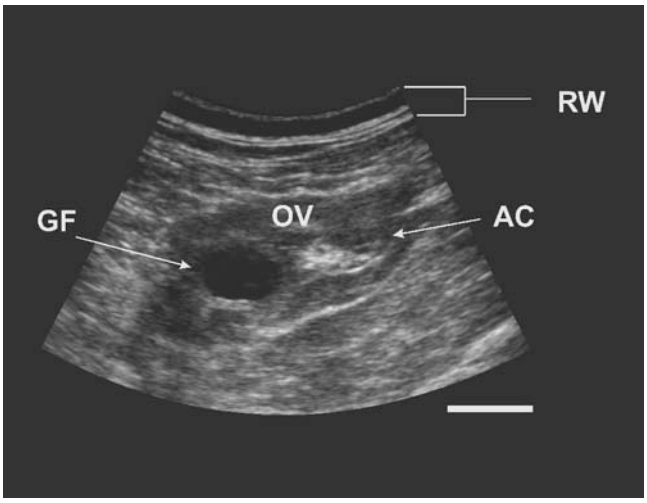


***Figure 27.36.*** *Transrectal sonogram (4-2) of an active ovary (OV) containing a Graafian follicle (GF) and an accessory corpus luteum (AC). The 20 mm follicle is already oval and will ovulate in the next 12 hours. The rectal wall (RW) appears as a moderate echoic strip on top of the sonogram.*

stalklike structures derived from the ligamentum located near the ovaries. However, they may confound the results of an US examination, being misinterpreted as follicular structures on the ovary; therefore, at least two US exams 1 week apart are necessary for clear differentiation of a paraovarian cyst from a follicular structure.

**Ovaries.** The ovaries in elephants are relatively small with dimensions of about 70 × 50  25 mm in adults. At 3–4 years of age, the ovaries develop a convoluted, brainlike surface that can be seen with US. Figure 27.35 shows a juvenile ovary in median-section, which is divided into the echogenic central *medulla ovarii* and the less echogenic peripheral *cortex ovarii*. In general, there are no large follicles or corpora lutea visible until the female enters puberty.

Two days before ovulation the active ovary contains a well-developed Graafian follicle (Fig. 27.36). Follicles are characterized sonographically by their round shape, anechogenic appearance, and typical white line below fluid-filled structures on the far side from the transducer in the viewing window. However, the distinction between ovarian follicles and fluid-filled cysts often is difficult, and subsequent US examinations are necessary to determine the true nature of the structure.[3]

Two to three days after conception a sonographically easily-distinguishable corpus luteum of pregnancy (Fig. 27.37) forms on the ovary at the site of ovulation. Typical corpora lutea derived from ovulation are large (>25 mm) and prominent on the ovarian cortex, in contrast to the smaller, intracortical accessory corpora lutea. In general, corpora lutea in elephants are moderately echogenic, with an elongated echogenic center and a homogenous parenchyma regardless of type. The total number of corpora lutea, including accessory corpora lutea, may range from 0–10 on each ovary, with higher numbers observed during middle and late gestation.[6] However, there generally appears to be only one large corpus luteum of pregnancy produced. Figure 27.38 shows part of an active ovary of a pregnant African elephant with a newly formed accessory corpus luteum containing a fluid-filled cavity 5 mm in diameter. These fluid-filled structures are rare, occurring in <5% of cows monitored, and they result from the luteinization of follicles, not ovulation. In general, differences in structure shape, location within the ovary, absence of stigmata, and, occasionally, the presence of a fluid-filled cavity

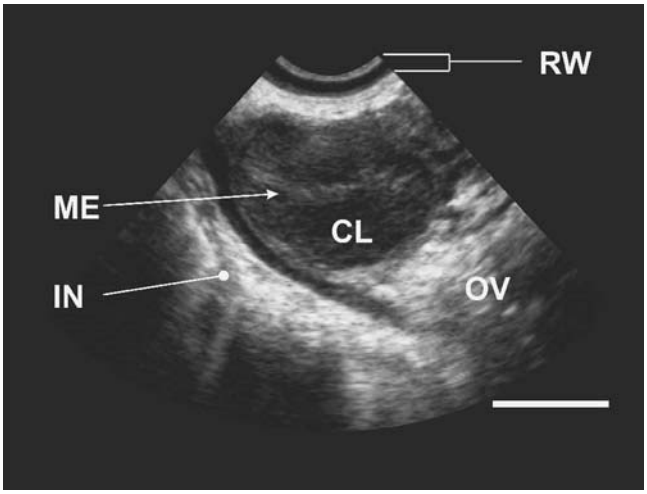


***Figure 27.37.*** *Transrectal sonogram (4-2 MHz) of a part of an ovary (OV) containing a large corpus luteum (CL) of pregnancy. The well-developed corpus luteum (CL) of pregnancy is characterized by a internal corpus fibrosum or mediastinum (ME). The ovary is located next to an intestinal loop (IN) filled with gas. The rectal wall (RW) appears as a moderate echoic strip on top of the sonogram.*

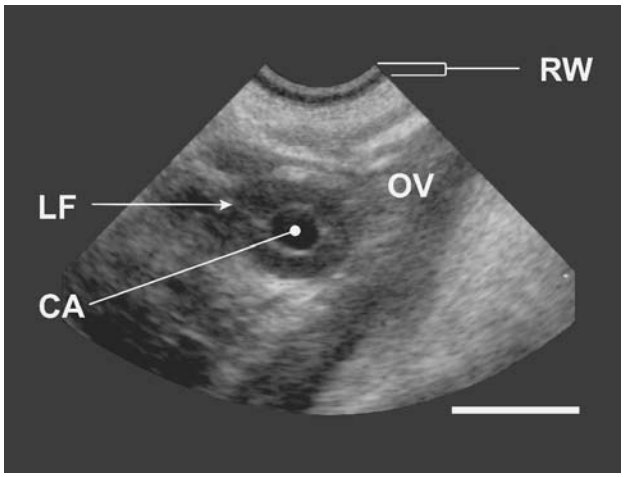


***Figure 27.38.*** *Transrectal sonogram (4-2 MHz) of an ovary (OV) containing a luteinized follicle (LF) with the typical internal cavity (CA). The rectal wall (RW) appears as a moderate echoic strip on top of the sonogram.*

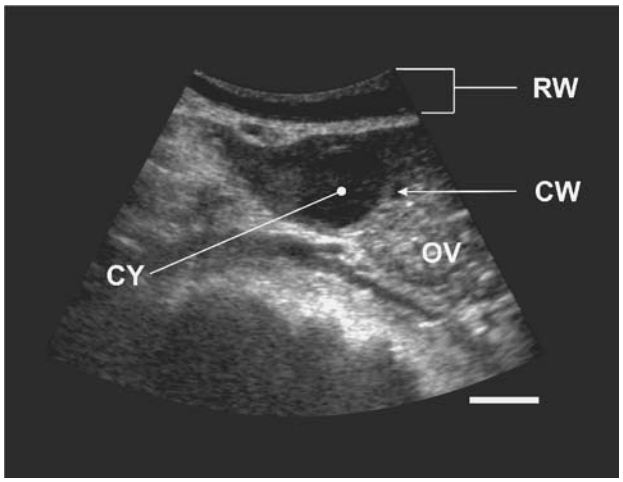


***Figure 27.39.*** *Transrectal sonogram (4-2 MHz) of an ovary (OV) containing an ovarian cyst (CY). The cyst can be clearly distinguished from a follicular structure by the prominent cystic wall (CW). Ovarian cysts are often combined with acyclicity in adult elephants. The rectal wall (RW) appears as a moderate echoic strip on top of the sonogram.*

distinguish accessory corpora lutea from corpora lutea produced after ovulation.

Minor pathologies of the ovary have been found in Asian and African elephants, such as cystic formations in the cortex up to 25 mm in diameter, characterized by a well-defined wall up to 2 mm thick. Small cysts appear as follicular structures located in the outer cortex as compared to pre-Graafian follicles, which are imbedded in the parenchyma. Large cysts are within the size range of normal Graafian follicles; however, the cyst wall is thicker than that of a normal follicle (Fig. 27.39). In general, ovarian cysts are observed in ~5% of captive Asian and ~15% of captive African elephants, but rarely are observed in free-ranging African females (<1%). At present, no data are available for wild Asian elephants. There are clear evidences that ovarian cysts are causing acyclicity based on coinciding blood sample analyses of progesterone and estrogens.[3]

In contrast to the relatively common acyclicity phenomenon in captive elephants characterized by an undetectable progesterone level, there has been only one identified case of a functional ovarian tumor that produced pregnancylike levels of progesterone for over 3 years before the primiparous Asian elephant died as a consequence of the tumor.

## CONCLUSIONS

This chapter provides credible applications of the value of ultrasonography as a practical diagnostic tool in elephants. The technology still is vastly underutilized, and its many advantages (noninvasiveness, reproducible real-time images, cross-sectional images of tissues/organs, and ability to measure morphometry) certainly argue for more widespread use.

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# 28 Reproductive Endocrinology

Janine L. Brown

## INTRODUCTION

Advances in endocrinology are contributing to an understanding of the complex mechanisms controlling normative reproductive function in the elephant. The ability to collect routine blood samples has provided a rare opportunity to study hormones in circulation, in many cases for decades. Endocrine function also can be assessed noninvasively through analysis of excreted steroid metabolites in urine and feces. Such findings are of interest from a scholarly perspective, but also have proven invaluable for breeding management, including development of artificial insemination techniques. Given the relative ease of sample collection, reasonable cost of analyses and critical need to increase birth rates within captive populations, it is imperative that elephants are part of a hormone monitoring program to aid basic research and applied management efforts.

## THE ESTROUS CYCLE

The elephant has the longest estrous cycle of any mammal studied to date, 13–17 weeks in duration with a 4–6 week follicular phase and an 8–10 week luteal phase.[41,75] A proposed endocrine model for the elephant estrous cycle is presented in Figure 28.1. A summary of normal hormone values for Asian and African females is given in Table 28.1.

### Progestins

Early endocrine studies found circulating progesterone concentrations were comparatively low in the elephant.[86,87,88] Subsequently it was determined that the major circulating luteal steroid is not progesterone, but 5α-reduced pregnanes.[39,45,46,47,85] In both species, 5α-pregnane-3, 20-dione (5αDHP) and 5α-pregnane-3-ol-20-one (5α-P-3-OH) are present in high concentrations, with 5αDHP predominating.[46,47] Recent demonstration of high affinity 5α-reduced progestin metabolite binding to elephant endometrial receptors[36,37,64] confirmed their biological significance, at least in African elephants. In the Asian elephant, 17α-hydroxyprogesterone (17α-OHP) also is present in circulation, albeit at lower concentrations.[47,71] The ability of "progesterone" assays to monitor luteal activity in the elephant is due to varying antibody cross-reactivities with circulating pregnanes. Assays using broad-spectrum antisera produce values that exceed those for "progesterone" by up to twentyfold.[47] However, data are valid if qualitative profiles are comparable, regardless of antisera cross-reactivity.

Progestins follow a biphasic pattern during the luteal phase, with concentrations gradually increasing to a midluteal peak and then dropping off more rapidly to baseline. Daily sampling has revealed a 1- to 2-day drop in progestins between days 2 and 9 of the luteal phase.[16] Most breeding activity occurs in the first few days of the luteal phase and ceases after the transient progestin drop.[16]

Progesterone, pregnanediol glucuronide, nor 20α-dihydroprogesterone are not excreted into urine or feces.[38,39,71,92] Rather, in the African elephant, 5α-DHP and 5α-P-3-OH are found in excreta as in blood, but with 5α-P-3-OH predominating.[26,39] Measurement of pregnanes in feces collected from free-ranging African elephants was diagnostic of pregnancy, with concentrations tending to be lower during the dry season after controlling for gestational stage.[28] Presumably 5α-DHP and 5α-P-3-OH are excreted in Asian elephant feces, but comparative studies are lacking. There is a species difference in the excretion of 5α-pregnanetriol, with the 17α-OHP metabolite being abundant in the urine[71] and feces[38] of Asian, but not African,[47] elephants. A new technique that combines headspace solid-phase microextraction (SPME) and gas chromatography–mass spectrometry (GC/MS) to measure urinary 5 Alpha-androst-2-en-17 Beta-ol has proven effective for characterizing luteal activity during the estrous cycle and pregnancy in Asian elephants.[22]

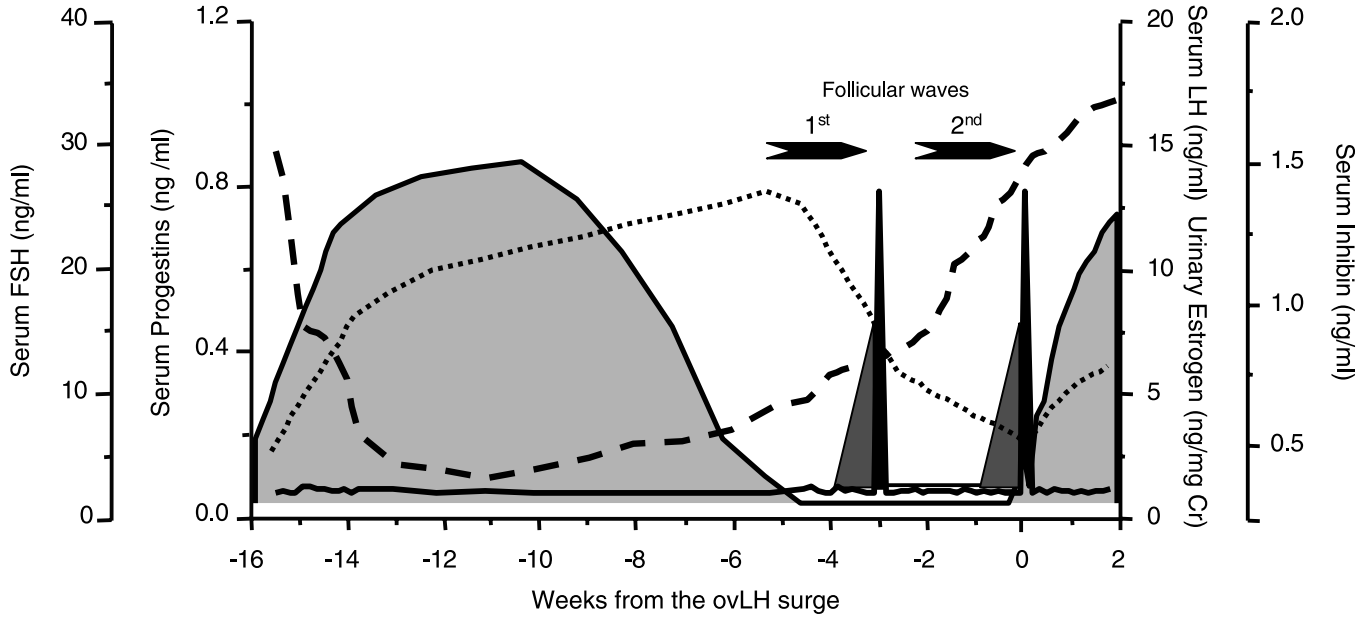


***Figure 28.1.*** *Proposed model of the ovarian cycle in the elephant. Elevated progestins (light stippled area) inhibit follicular development and LH secretion during the luteal phase. High FSH concentrations (dotted line) at the beginning of the nonluteal phase recruit follicles and initiate two successive waves of follicular development that culminate in two distinct LH surges (solid line) occurring 3 weeks apart. Estrogens (dark stippled area) increase before each LH surge as follicles develop. The first follicular wave consists of multiple follicles that do not reach Graafian size or ovulate, but regress after the anLH surge. Over the next 3 weeks, a second follicular wave results in the selection of one large dominant follicle that ovulates about 24 h after the ovLH surge. The slight drop in progestins during the first few days of the luteal phase may represent a shift in steroidogenic activity between accessory luteal structures and the newly formed postovulatory corpus luteum, or follicle luteinization. Thereafter, progestins increase in conjunction with continued accessory luteal and corpus luteum maturation, followed by the gradual rise in FSH, which peaks at the end of the luteal phase. Inhibin concentrations (dashed line) follow an inverse pattern to that of FSH.*

## Estrogens

To date, measurements of circulating free estradiol have not reliably reflected follicular activity.[12,47,73] Serum concentrations are low (<10 pg/ml), presumably due to the high blood to Graafian follicle fluid volume ratio, which is five- to twentyfold greater than that of domestic mammals. Estradiol also is quickly metabolized to estrone and estradiol conjugates in the bloodstream,[19] so traditional free steroid extraction methods would not yield reliable results. Conjugated metabolites are excreted primarily in urine (95%),[19,92] explaining the futility of fecal estrogen analyses to assess follicular activity.[92] Recently, a sensitive urinary estradiol-3-glucuronide assay identified two waves of follicular steroidogenic activity in Asian elephants[20] that agreed with ultrasound data in African females.[40] Similar ultrasound studies in Asian elephants and urinary estrogen analyses in African elephants have not been conducted. An increase in urinary estrogen conjugates also is observed during the luteal phase,[20,61] suggesting that the corpus luteum may be a source of estrogens in the elephant, as it is in primates.[77]

## Pituitary Hormones

A number of reproductive hormones (prolactin, LH, FSH) have been isolated from elephant pituitary glands and appear functionally similar to those of humans and domestic mammals.[55,56,62] Partially purified elephant pituitary preparations have been used to validate heterologous assays based on combinations of human, ovine, bovine, and equine components.[4,6,62,63]

Ovulation in most mammals is induced by a single, preovulatory LH surge. By contrast, daily sampling during the follicular phase has revealed that the elephant exhibits two LH surges, referred to as the *double LH surge*.[10,51] The first surge occurs 10–20 days after the drop in progestins, with the second occurring 19–22 days later. The surges are quantitatively and qualitatively similar, but only the second induces ovulation. The terms *anovulatory LH (anLH)* and *ovulatory LH (ovLH)* are used to define these surges, which follow each of two functionally distinct follicular waves.[40] Multiple (2–4) small follicles, none of which ovulate, develop during the first wave, whereas only one large antral follicle becomes dominant and ovulates during the second wave. Ovulation occurs ~24 hours after the ovLH surge.[14] In both species, progestins normally rise 1–3 days before the ovLH surge.[10,51] One speculation for the preovulatory progestin rise is that nonovulatory follicles of the first wave form accessory corpora lutea and produce progestins necessary for ovulation of the subsequent Graafian follicle.[40] It is also possible that preovulatory follicle luteinization may be occurring. Compara-

**Table 28.1.** Overall Mean (+SEM) and Normal Mean Range Concentrations of Baseline Serum Pituitary, Ovarian, Thyroid, and Adrenal Hormones in Asian and African Elephant Females and Males

| | Female | | Male | |
|---|---|---|---|---|
| | Asian | African | Asian | African |
| LH[1,2,3,4] | 0.82 ± 0.06 | 0.67 ± 0.05 | 0.68 ± 0.07 | 0.71 ± 0.09 |
| (ng/ml) | (0.25–1.02) | (0.34–0.98) | (0.38–1.11) | (0.36–1.15) |
| FSH[1,3,4] | 4.3 ± 0.3 | 4.4 ± 0.3 | 2.3 ± 0.4 | 2.7 ± 0.3 |
| (ng/ml) | (1.9–6.4) | (0.6–6.4) | (0.9–3.4) | (1.0–3.3) |
| Prolactin[1,3,4] | 4.9 ± 0.4 | 7.8 ± 0.5 | 2.8 ± 0.3 | 2.5 ± 0.3 |
| (ng/ml) | (2.3–6.8) | (4.3–8.8) | (.9–6.8) | (1.0–7.0) |
| TSH[1,3] | 0.75 ± 0.10 | 0.62 ± 0.08 | 0.63 ± 0.02 | 0.56 ± 0.06 |
| (ng/ml) | (0.61–1.08) | (0.4–1.3) | (0.26–0.99) | (0.28–1.09) |
| Inhibin[5] | 0.75 ± 0.24 | ND | ND | ND |
| (ng/ml) | (0.13–1.01) | | | |
| Estradiol[4] | 14.7 ± 2.3 | 24.9 ± 6.1 | ND | ND |
| (pg/ml) | (13.9–15.5) | (14.3–42.3) | | |
| Free T3[1,3] | 1.9 ± 0.3 | 1.6 ± 0.3 | 1.8 ± 0.3 | 2.2 ± 0.3 |
| (pg/ml) | (1.1–2.9) | (0.7–3.5) | (0.4–3.4) | (0.9–6.1) |
| Free T4[1,3] | 1.01 ± 0.06 | 0.91 ± 0.03 | 0.64 ± 0.05 | 0.76 ± 0.01 |
| (ng/dl) | (0.74–1.44) | (0.72–1.11) | (0.42–1.31) | (0.32–1.04) |
| Total T3[1,3] | 123.9 ± 6.3 | 124.0 ± 4.3 | 112.1 ± 0.8 | 106.2 ± 1.3 |
| (ng/dl) | (91.4–158.4) | (99.3–148.1) | (64.8–189.4) | (77.8–128.1) |
| Total T4[1,3] | 11.2 ± 0.6 | 10.1 ± 0.4 | 8.67 ± 0.12 | 9.9 ± 0.7 |
| (g/dl) | (8.6–14.5) | (7.6–12.2) | (5.8–18.0) | (5.7–12.7) |
| Cortisol[1,3] | 23.3 ± 4.2 | 20.4 ± 4.8 | 14.4 ± 2.4 | 17.9 ± 2.9 |
| (ng/ml) | (11.1–51.6) | (5.7–59.6) | (4.6–98.7) | (9.2–59.9) |
| DHT[6] | 0.02 ± .01 | 0.22 ± 0.05 | 0.54 ± 0.29 | 0.22 ± 0.04 |
| (ng/ml) | (0.01–0.03) | (0.15–0.37) | (0.16–1.52) | (0.15–0.30) |
| Testosterone[6] | 0.08 ± 0.01 | 0.25 ± 0.08 | 7.7 ± 1.1 | 7.6 ± 0.6 |
| (ng/ml) | (0.05–0.11) | (0.04–0.46) | (0.1–29.4) | (0.2–19.7) |

ND = Not Determined.
[1]Bull data are unpublished.
[2]Excludes LH surge data.
[3]Brown 2004a.
[4]Brown 1999a.
[5]Brown 1991.
[6]Rasmussen 1984, Cooper 1990, Lincoln 1996.

tive analyses indicate that on average the anLH and ovLH surge concentrations are higher in Asian (5–30 ng/ml) than African (1.5–8 ng/ml) females. Significant LH surges are rarely observed during the luteal phase.[12]

**Follicle stimulating hormone (FSH).** FSH has a protracted secretory profile that lags behind progestin changes by about a week. In both species, FSH is highest at the end of the luteal phase and decreases during the follicular phase, reaching baseline concentrations just before the ovLH surge.[6,10,13] This profile differs somewhat from other monovular species where FSH increases only after removal of the progestin block. In general, the function of FSH is to stimulate follicular development, including activation of granulosa cell aromatase and estrogen production. Elevated peripheral estrogens and inhibin then suppress FSH so that circulating concentrations fall below that needed to stimulate maturation of less-developed follicles. A decline in FSH at the end of the follicular phase permits dominant follicle selection, and an increase in estradiol elicits an ovulatory LH surge. Smaller follicles fail to reach a similar developmental stage because they remain FSH-dependent, and those concentrations are too low to provoke further growth.[29,34,97] In the elephant, none of the follicles during the first wave achieves this maturation capability. It is possible that FSH concentrations early in the follicular phase are too high to facilitate dominant follicle selection. As the follicular phase progresses, increases in estrogens[20] and inhibin[6] presumably suppress FSH. It is only after FSH concentrations decline toward baseline that a second wave results in dominant follicle selection and ovulation.

The purpose of the two follicular waves and precisely timed LH surges is unclear, but they may have an evolutionary purpose. Given the long distances bulls travel in search of estrous females in the wild, it would be beneficial for females to announce impending fertility. Elephants sometimes exhibit a "false behavioral estrus"[54] several weeks before true estrus, which may be related to excretion of a urinary pheromone, (Z)-7-dodecenyl acetate (Z7-12:Ac), described in Asian elephants.[21,80] Concentrations increase in conjunction with the first follicular wave, although peak Z7-12:Ac

levels are not attained until the ovLH surge. A comparable pheromone has yet to be identified in African elephants. From a practical standpoint, the double LH surge is useful for timing breeding to coincide with ovulation by scheduling natural mating or artificial insemination 3 weeks after the first anLH surge.[14]

**Prolactin.** A notable species difference exists for prolactin secretion; concentrations increase during the follicular phase in African,[4,13] but not in Asian,[9,10,13,16] elephants. Thus, overall average prolactin concentrations are higher in African than Asian females. In other species, prolactin increases in conjunction with follicular development.[17] Thus, it may be of reproductive significance, at least in the African species. Prolactin secretion is controlled by a negative feedback action of dopamine on pituitary lactotrophs. In our laboratory, we have shown that prolactin secretion in the elephant is stimulated by dopamine antagonists such as domperidone and inhibited by dopamine antagonists such as cabergoline.[3,65]

## PREGNANCY

Patterns of serum progestins, prolactin, and relaxin in pregnant Asian and African elephants are depicted in Figure 28.2. Pregnancy lasts 20–22 months and is diagnosed by elevated progestins beyond the normal luteal phase (after about week 12). Postconception, the progestin pattern for the first 8 weeks is similar to a nonpregnant luteal phase. Concentrations decline by about half before increasing to levels that generally exceed those in nonpregnant females.[8,41,63,74] Concentrations overlap enough, however, that serial samples are required for accurate diagnosis. Transitory decreases in progestin secretion occur at around 2 and 12 months, which may signal shifts in luteal or placental steroidogenic activity. Based on histological examination[88] and analysis of luteal progestins,[46] the corpus luteum of pregnancy is most steroidogenically active between 2 and 14 months of gestation.[47] In the early weeks of gestation (weeks 2–7), a change in the 17 Alpha-hydroxyprogesterone (OHP):progesterone ratio occurs in Asian elephants, with a ratio of $\leq$0.7 indicating pregnancy.[72] Whether this hormonal shift occurs in African elephants, or is valid using other "progesterone" assays with differing antisera specificities remains to be determined. Human pregnancy test kits do not work because elephants do not produce a pregnancy protein that cross-reacts with hCG.

The source of gestational progestins is unknown, but the lack of a correlation between circulating progestin levels and luteal volume,[24] the histological appearance of luteal tissue,[88] and a positive relationship between fetal progestin concentrations and gestational age[24] suggest the placenta may produce at least some.[47] However, incubation of placental tissue from African elephants with tritiated steroid precursors yielded no labeled progestins, nor was there any specific immunochemical staining for enzymes involved in the steroidogenic pathway.[2] Uterine receptor studies in African elephants found progesterone receptor concentrations were inversely related to circulating pregnanes and decreased as a function of gestational age.[36,37] Comparable uterine and placental studies have not been conducted in the Asian elephant.

There is a species difference in progestin secretory patterns during gestation. In African elephants, progestin concentrations are higher during the first half of gestation and then decline, often dramatically, at midgestation and remain lower than those in Asian elephants until parturition.[66] In Asian elephants, there is a difference in maternal progestins related to fetal gender, with concentrations being higher in females carrying male calves.[66] This was not observed in African elephants. Fetal sex differences in maternal androgens also have been reported in Asian[25] and African (Kiso, Schmitt, Carden, unpublished) elephants, with higher circulating testosterone in female elephants carrying male fetuses.[25] Duer suggested the elevated testosterone might be of fetal testicular origin, although the elephant corpus luteum apparently also produces androgens.[81,90] The fetal sex difference in progestins could be related to testicular steroid production, given that in other species progesterone produced by fetal Leydig cells is converted to testosterone to complete male duct system development. It should be noted that species and fetal gender differences in progestins are significant using antibodies with differing cross-reactivities for circulating pregnanes.[66]

Prolactin immunoreactivity increases markedly after 5–7 months of gestation in both Asian and African elephants,[8,9,44,63,66] permitting pregnancy diagnosis based on single sample analyses. Given the magnitude of the increase (twenty- to hundredfold), at least some of the immunoreactivity is probably due to antibody cross-reactivity with placental lactogens. These may be important for sustaining corpus luteum activity, stimulating fetal growth, and/or aiding in preparation of the mammary glands for lactation, as in other species.[58] Concentrations of free estrogens in serum are low and unchanging during gestation,[41,43] whereas circulating conjugated estrogens increase significantly during the latter half in Asian and African elephants.[43,44] Conjugated estrone tends to be higher than estradiol during the early stages of pregnancy, but then at about 6 months the ratio shifts in favor of conjugated estradiol.[43] Interestingly, the shift occurs about the time serum prolactin immunoreactivity is increasing.[8,9,44,63,66] Thus, this relationship may be significant given the ability of estrogens to stimulate prolactin.[53] In African elephants, total estrogens in hydrolyzed urine increase after 30 weeks, peak at midgestation and decline until birth. By contrast, urinary estrogens are elevated throughout ges-

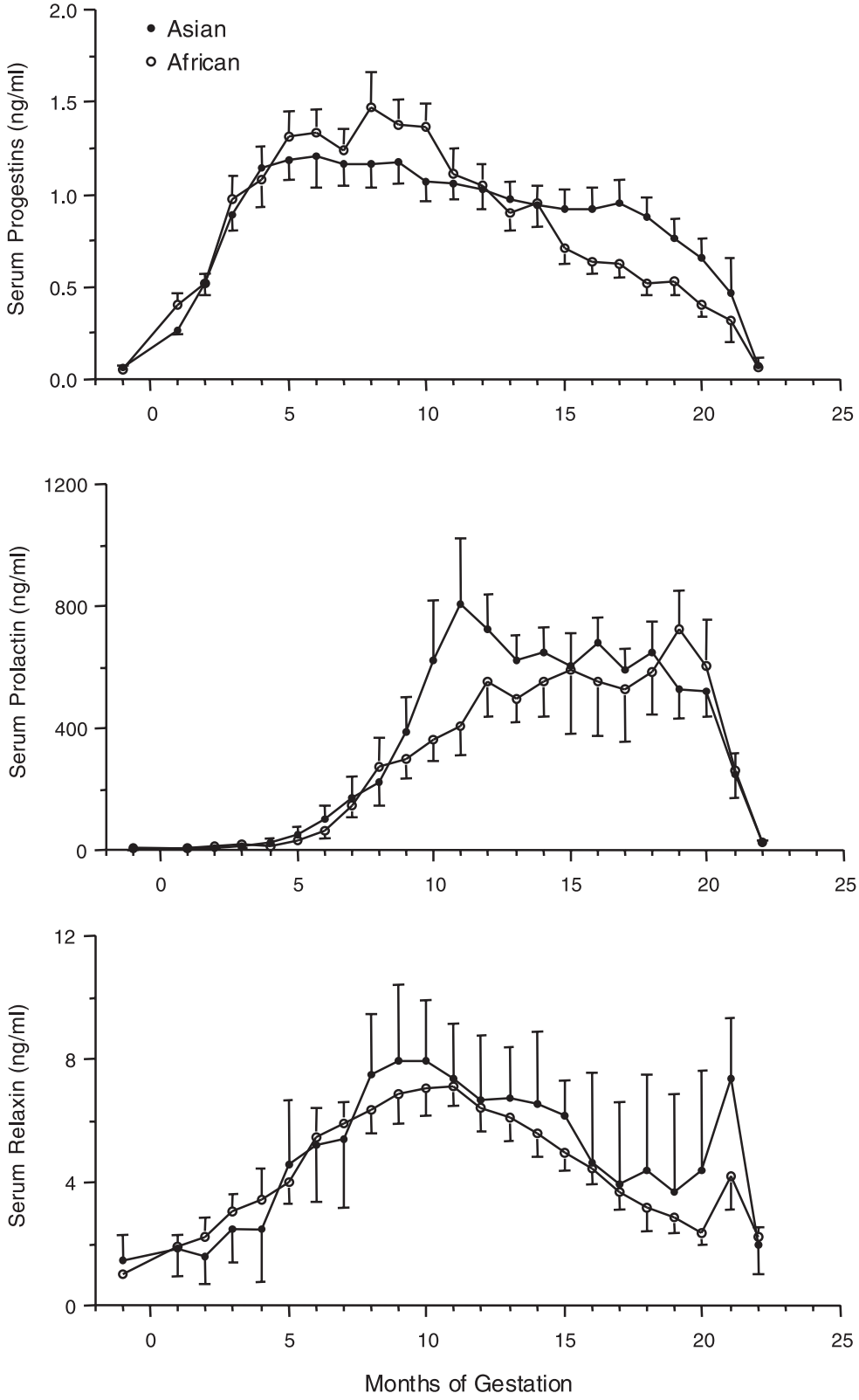


***Figure 28.2.*** *Profiles of serum progestins, prolactin, and relaxin throughout gestation in Asian and African elephants. Decreases in progestins at ~2.5 and 13 months of gestation signal possible shifts in luteal/placental function. Prolactin increases significantly after the fifth month of gestation and is a reliable tool for diagnosing pregnancy in single samples. Relaxin surges at the end of gestation to aid in parturition. Birth normally occurs 2–5 days after progestins decline to baseline (from Brown 2004c).*

tation in the Asian elephant.[61] The source of gestational estrogens is not known, but Hodges[47] and Fieß[26] suggest that the placenta and/or fetal gonads may play a role in estrogen production.

Not surprisingly, measurement of fecal estrogens during pregnancy is of limited value, although a modest increase in total estrone was observed after midgestation in African elephants.[26] This contrasted with urinary estrogen data where concentrations declined after midgestation. The authors concluded that an increase

in fecal estrone levels in the face of falling urinary estrogens may indicate a shift in metabolism and/or excretion, at least in African elephants.[26] Comparable studies of fecal estrogens in Asian elephants during gestation are lacking.

Serum relaxin increases for the first 10 months and then declines until a few weeks before birth when a sharp rise occurs.[66,73] Relaxin probably plays a role in parturition similar to that in other species by facilitating a softening of the cervix and loosening of pelvic ligaments, and ensuring synchrony in uterine muscles after labor begins.[73]

There is no evidence of a placental gonadotropinlike factor in the elephant.[2,12] Assays for eCG, hCG, and Pregnancy Specific Protein B failed to detect immunoreactivity in serum of pregnant Asian or African elephants.[12] This lack of immunoactivity means that commercial pregnancy detection kits would not be effective in elephants. Allen[1] also reported no gonadotropin bioactivity in placental extracts from early pregnancy to midpregnancy in African elephants. However, the formation of new follicles and fresh corpora lutea at 4 to 5 months postconception in African elephants[42] suggests further studies are needed to better understand postconception endocrine function.

Serum cortisol secretion is stable throughout gestation until 1–2 weeks before birth when one or more discrete spikes occur.[66] These are suspected of being part of an endocrine cascade that facilitates parturition, similar to that described for other species.[35,52,82] A surge in cortisol in serum and/or urine around the day of parturition presumably is related to the "stress" of active labor and birth.[8,66] By contrast, Foley[28] reported that fecal corticoids tracked progestin excretory profiles throughout gestation in free-ranging African elephants. However, seasonal changes in corticoids also were observed and it was not clear if this was factored into their analysis.

Progestins drop to baseline 2–5 days before parturition,[8,16,21,26] providing adequate time to prepare for birth. Lactational anestrus lasts from 8–12 months, although retained placenta, death of a calf, or premature weaning can reduce the postpartum period to as little as 8 weeks.[8,74]

## REPRODUCTIVE ENDOCRINE PROBLEMS

Some females exhibit stable, baseline concentrations of serum progestins indicative of ovarian inactivity, called *flatliners*. Up to 14% of Asian and 29% of African elephants in North America exhibit this problem at any given time.[15] Similar statistics have been reported for elephants in Europe (A.-K. Oerke, unpublished). In Asian elephants, most noncycling females are older (>30 years of age), whereas in African elephants acyclicity is distributed across age classes.[15] Ovarian status is a dynamic process, however. There are numerous examples of cycling elephants that suddenly stop for prolonged periods and noncycling females that resume cyclicity and even have conceived. Other elephants exhibit irregular cyclicity, usually in the form of prolonged nonluteal periods.[15,83]

The probability that the etiology of irregular or acyclic ovarian activity is the same for all females is unlikely. In one group of three female African elephants, Schulte[83] found temporary ovarian inactivity was related to an increase in time spent indoors during winter months. Freeman[30] reported that African elephant females rated by keepers as dominant were more likely to be acyclic than subordinate herdmates. Ovarian follicular cysts have also been associated with acyclicity.[11,15] One comprehensive endocrine study revealed that average prolactin concentrations were significantly higher (range, 15–50 ng/ml) in a third of noncycling females, all African but one.[15] Hyperprolactinemia is a cause of infertility in women and livestock and causes are diverse, but often it is associated with prolactin-secreting pituitary tumors.[50] Hypothalamic dopamine regulates prolactin through an inhibitory mechanism, so any lesion interfering with its synthesis, release or activity can affect prolactin secretion.[59,60,95,96] Transient increases in prolactin may be caused by sleep, protein meals, and hypoglycemia.[50,95,96] These are not likely causes of elevated prolactin in acyclic elephants, however, because the condition is chronic, not transient. Hyperprolactinemia induced by physiological or psychological stressors has been linked to increased adrenocorticotropin hormone and cortisol secretion,[27] and it is well known that stress-activated cortisol can inhibit reproduction.[69] However, although four of five elephants with significantly elevated mean cortisol were flatliners, they represented only a small proportion of the total acyclic group.[15] Hypothyroidism (i.e., reduced T4) can cause hyperprolactinemia through an increase in TRH that stimulates TSH and prolactin.[50,95,96] But so far there has been no evidence of altered thyroid function (based on TSH, free and total T3 and T4) in acyclic elephants, with the exception of one overweight acyclic Asian female that had elevated TSH.[15] Treatment of a hyperprolactinemic Asian elephant with the dopamine agonist, cabergoline, reduced prolactin to baseline concentrations and reinitiated ovarian cyclicity.[3,66] Trials to restore reproductive cyclicity by reducing chronically elevated prolactin levels in other elephants currently are underway.

## ENDOCRINE FUNCTION IN MALES

The sexually active period in bull elephants is called *musth,* and is characterized by heightened aggressive and sexual behavior, temporal gland drainage, urine dribbling, and increased androgen secretion for periods of a few weeks to several months (see reviews, Schmidt,[84] Mikota,[67] Niemuller[73]). A summary of normal hormone values for bull elephants is presented in Table 28.1.

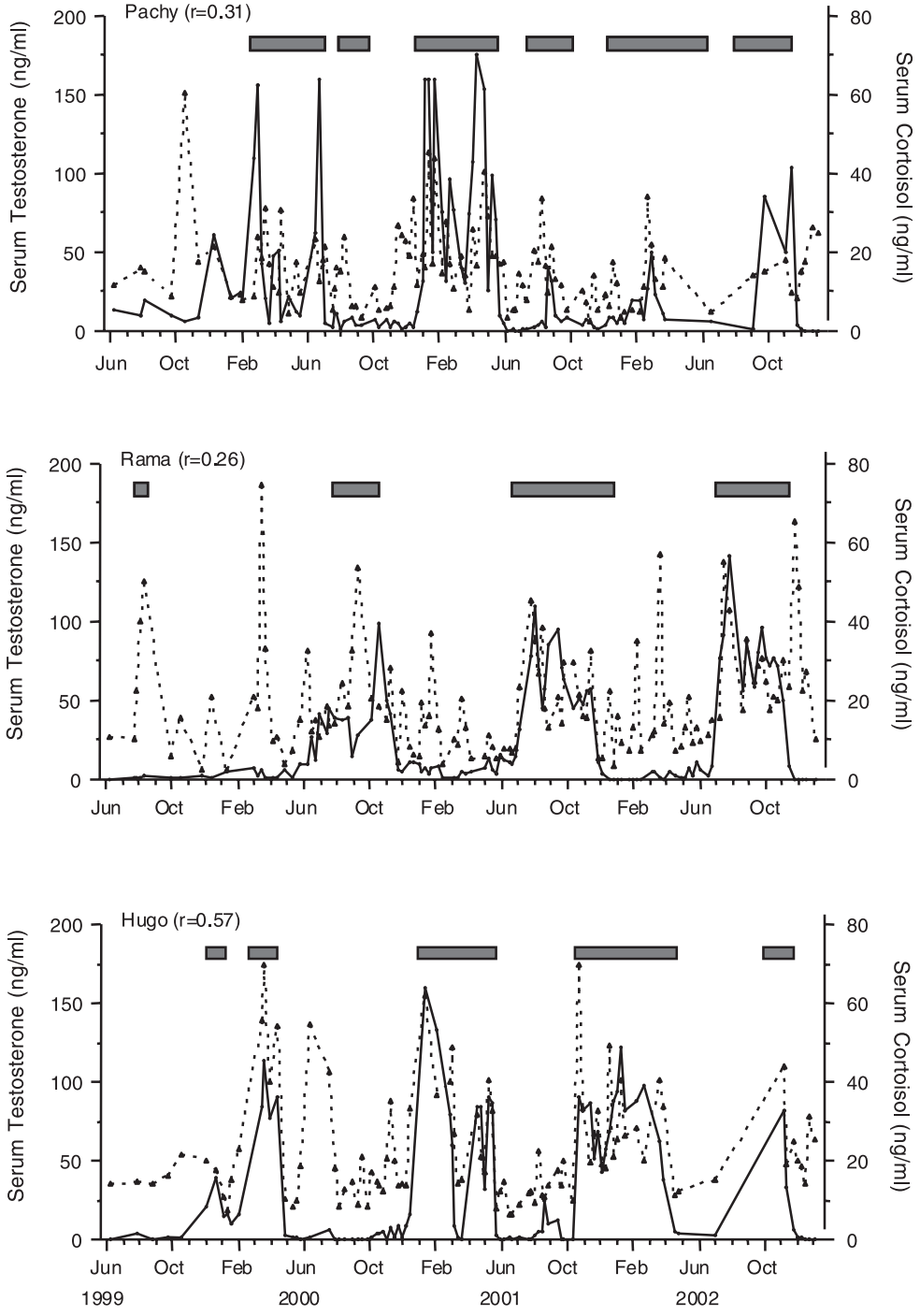


***Figure 28.3.*** *Profiles of serum testosterone (solid line) and cortisol (dashed line) in Asian elephant bulls at the Oregon Zoo. Numbers in parentheses are the correlation coefficients for the relationships between testosterone and cortisol. Solid bars represent periods of musth.*

## Androgens

Testosterone production is age dependent and related to social rank, with dominant males exhibiting higher average testosterone concentrations than subordinates.[57] Generally, testosterone concentrations in nonmusth males average less than 5 ng/ml, but they can exceed 100 ng/ml in full musth (Fig. 28.3).

During periods of nonmusth in the Asian elephant, circulating androstenedione concentrations are greater than testosterone, whereas the ratio switches in favor of testosterone during musth.[70] Dihydrotestosterone increases during musth in parallel with testosterone, although not to the same extent.[79] The temporal glands concentrate testosterone and DHT four- to tenfold over blood levels at all reproductive stages in African males and females, but only during musth in Asian bulls.[79]

Radiolabel infusion of $^{14}$C-testosterone indicated that 57% of radioactivity was excreted in feces and 43% in urine.[32] Most radioactive metabolites in urine were conjugated (97%) and enzyme hydrolyzable (78%),

whereas in feces nearly all (>99%) were unconjugated steroids. Urine excretion occurred within hours of injection, whereas peak fecal radioactivity was observed ~1.5 days later. HPLC analyses identified immunoreactivity associated with native testosterone, androstenedione, and epiandrosterone in urine and feces; feces also contained a metabolite that coeluted with androsterone.[31] Poole[76] reported finding testosterone and DHT immunoreactivity in urine HPLC fractions. Measurements of immunoreactive androgen metabolites using assays for testosterone, androstenedione, or epiandrosterone are effective for assessing testicular activity noninvasively in captive and free-ranging African bull elephants.[5,31,32,33] Similar studies on androgen metabolism and excretion in Asian bulls are lacking.

## Pituitary Hormones and GnRH Analogs

Androgen secretion in elephants is under LH control, with pulses of testosterone closely following those of LH (~1 pulse/3 hours).[70] There is little difference in LH pulse rate between musth and nonmusth Asian bulls; however, pulse amplitude and area are greater during musth.[70] Mean LH concentrations increase about 4 weeks before musth begins, declining to baseline levels soon thereafter.[91] In both species, testes exhibit a hyperresponsiveness during musth, with greater testosterone excreted after LH pulses and in response to GnRH challenge.[7,57,70] In the absence of testosterone in castrates or hypogonadal males, mean basal gonadotropins are elevated (LH, 3.5–8.9 ng/ml; FSH, 3.4–9.6 ng/ml), similar to that in other mammals.

Inhibiting pituitary control of testicular steroidogenic activity might attenuate musth signs. Treatment with GnRH analogs (antagonist, Detirelix [Syntex Research, Palo Alto, California, USA]; agonist, Leuprolide Depot [Takeda-Abbott, Abbott Park, Illinois, USA]) temporarily suppressed serum LH and testosterone secretion in free-ranging African elephants.[7] A single injection of Leuprolide in a captive musth bull also reduced testosterone to castrate levels for 2 months, but did not completely eliminate behavioral signs.[68] In another study, it took several years of Leuprolide treatment to effectively suppress all aspects of musth in a captive Asian bull.[23]

## Corticoids

Cortisol secretion follows a diurnal pattern in male and female Asian and African elephants (Fig. 28.4), similar to other species. Following radiolabel infusion of $^{3}$H-cortisol into an African bull, 82% of metabolites were excreted into urine and 15% into feces.[32] Almost all radioactive metabolites in urine were conjugated (86%) and enzyme hydrolyzable (53%), whereas most metabolites (86%) in feces were unconjugated. HPLC analyses identified at least five peaks of radioactivity in urine, one of which corresponded to native cortisol. There were three main radioactive peaks in feces, none of which were associated with cortisol. Corticoid metabolite excretory lag times of hours for urine and ~1.5 days for feces are similar to those reported for testosterone[32] and ovarian[92] steroids. Gainswindt[32] tested several antibodies against 3 Alpha,11-oxo-cortisol metabolites and found one against 11-oxo-etiocholanolone that had minimal cross-reactivity with excreted C-testosterone[14] metabolites in African bulls and was effective in characterizing corticoid excretion in urine and feces.[32] A commercial corticosterone RIA also has been validated for assessing adrenal activity in female African elephants,[28,93] but its suitability for bull elephants (i.e., lack of androgen metabolite cross-reactivity) has not been established. Studies also are needed to characterize corticoid metabolism and excretory products similarly in Asian bulls.

There is disagreement as to whether musth is driven

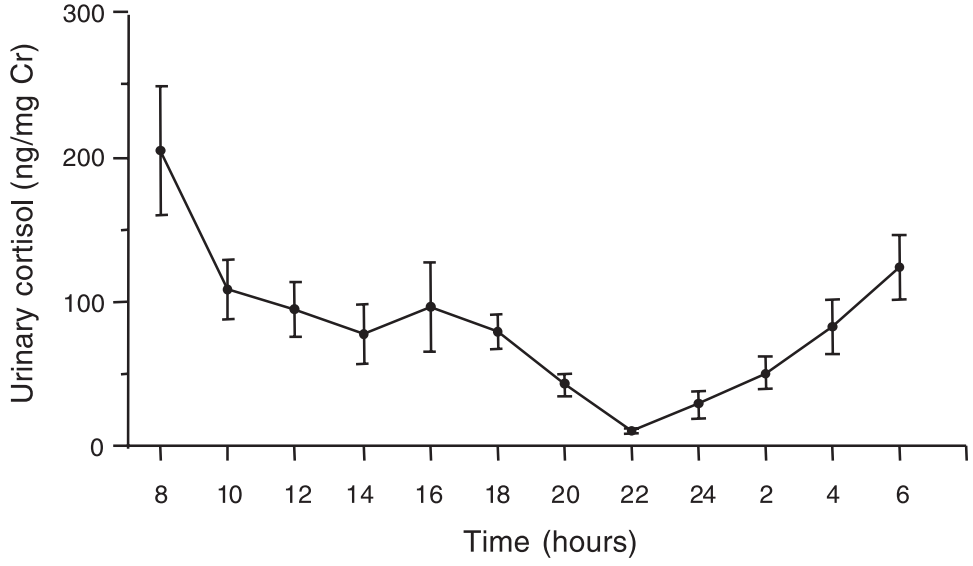


***Figure 28.4.*** *Mean (±SEM) concentrations of urinary cortisol showing a diurnal excretory pattern in Asian and African elephants (n = 6) (Wagener and Brown, unpublished).*

by testicular androgens alone or if it may be related, in part, to changes in adrenal activity. Noninvasive analyses (urine and/or feces) found no clear musth-related increases in corticoid metabolite excretion in captive[32] or free-ranging[33] African bulls. By contrast, a preliminary analysis of serum cortisol found elevations during musth periods in captive Asian and African elephants.[94] A study in our laboratory also identified modest but significant correlations between serum cortisol and testosterone in several captive Asian and African bulls (10 of 12) (average r = 0.33; range, 0.23–0.52) (Fig. 28.3). Serum cortisol secretion tends to be more variable than testosterone, with surges occurring in bulls in and out of musth (Fig. 28.3). More studies are needed to determine why circulating and excreted corticoid data appear to differ with respect to musth status in sexually mature elephant bulls.

## CONCLUSIONS

Endocrine monitoring is the key to assessing reproductive status of elephants, and it can be done using a variety of assay techniques (RIA, EIA, SPME/GC-MS). Profiles may be generated using serum, plasma, urine, or feces. Still, although we now have a fairly broad understanding of elephant endocrinology, a number of information gaps still exist. More work is needed to better understand the following: 1) cause and function of the double LH surge, 2) endocrine control of pregnancy, 3) cause of ovarian acyclicity, and 4) endocrine control of musth.

Endocrine treatments are needed to mitigate infertility problems in males and females and control the estrous cycle in females for facilitating breeding efforts. Future studies must be multifaceted, focusing on research to improve basic knowledge while using technology to support in situ and ex situ conservation projects. Last, it is important to take care in extrapolating data across species. Although there are many similarities in endocrine function between Asian and African elephants, there are enough differences to warrant conducting comparative studies on all aspects of reproductive biology.

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# 29 Urinary System

R. Eric Miller

## UNIQUE ANATOMY

The elephant kidneys are lobulated and contain 8 (±2) lobes (although one report notes 12 lobules).[6,7,8] The left kidney is farther caudal than the right.[7] In the male, the testes are located on the caudal aspect of the kidneys.

Each kidney contains approximately $15 \times 10^5$ glomeruli, and the mean glomerular diameter is 311 µm. The cortex is approximately 73% of the renal mass, and the medulla is 27%. Each kidney is supplied by one renal artery that branches at the renal hilus. The renal tubules are notable for hairpin loops in the cortical area, a situation that resembles that of the renal structure of the manatee, but not that of the dugong.[7] There are few differences in the anatomy of the kidneys, ureters, and bladder between the Asian and African elephant.

The urethra enters the urogenital canal of the female just posterior to the junction of the vagina and the urogenital canal. Its opening is characterized by a small papilla.[1,2] The urogenital canal extends 40–100 cm from the vaginal junction to the vulva.[16]

## UNIQUE PHYSIOLOGY

A typical elephant urine void is 5–11 liters (l), and the average daily is 50 l.[19] Correspondingly, elephants drink 140–200 l of water/day, obviously with variations based on body size and ambient temperature.[16,17] The color of normal urine varies from clear to straw-colored to cloudy. Calcium oxalate crystals (and others, notably calcium carbonate) are often seen in normal urine and their absence may indicate disease.[9,15] Specific gravity ranges from 1.004–1.033 (1.018 mean) and the urine is nearly always alkaline (range 6.8–8.0).[3,19] Daily urine void includes 2 g of solids (20% mineral and 80% organic content).

Uric acid appears in elephant urine, and some have speculated that its production may be endogenous because elephants do not eat foods high in purines (e.g., meat).[18]

A typical urinalysis is illustrated in Table 29.1.

Elephants typically have low serum Blood Urea Nitrogen (BUN) values. Table 29.2 illustrates the normal values for sera BUN and creatinine values in Asian and African elephants.

## DIAGNOSTIC TECHNIQUES

Indicators of renal disease are similar to those in other species. Changes in the urinalysis may indicate infection (leukocytes, proteinaceous or granular casts, proteinuria) or decreased ability to concentrate urine. Isosthenuria has been noted in elephants with both acute (e.g., pyelonephritis), and chronic, end-stage renal disease. Hemogram findings include leukocytosis in infections and an elevated BUN and creatinine when kidney function is compromised. Some have suggested that the disappearance of calcium oxalate crystals from normal urine may serve as an indicator of abnormalities, and conversely, a return to normal function after treatment.[9,15] It had also been suggested that elevated serum γ-glutamyl transferase (GGT) may indicate acute renal disease, whereas GGT levels may be normal in chronic disease.[9]

At least one author felt that fractional excretion rates of electrolytes were useful and illustrated that rates of sodium, chloride, and potassium levels in an Asian elephant with chronic renal disease were notably elevated when compared to three normal elephants.[9]

Ultrasound scans of the kidney demonstrate that the interlobar septum, calyx, and secondary infundibulum are hyperechoic, and that the cortex is more echogenic than the medulla.[13] Rectal ultrasound has been used to evaluate elephant kidneys postpyelonephritis[15] and to allow for the performance of ultrasound-guided biopsies in a juvenile (8-year-old) African elephant.[4] In the latter case, a 133 cm, 8-gauge tissue biopsy needle was inserted in the left paralumbar fossa.[4]

The urethra has been examined endoscopically, although "with difficulty."[14]

**Table 29.1.** Elephant Urinalysis

| Parameter/Unit | Ruedi 1995 | Benedict 1938 | Range (Average) Simon 1959 |
|---|---|---|---|
| SG | 1.010–1.036 | 1.012–1.027 (1.018) | 1.022–1.032 (1.026) |
| pH | 6.8–8.6 | All alkaline | |
| Na (mEq/L) | 1 | | |
| K (mEq/L) | 160–172 | | |
| Ca (mg/dl) | 72–244 | | |
| Mg (mg/dl) | 92.4–119.1 | | |
| P (mg/dl) | 0.12–0.248 | | |
| Glucose (mg/dl) | 2.52–6.30 | Negative | 2/38 glucose positive, 4 trace glucose |
| Creatinine (mg/dl) | 113–203.6 | | 32.3–238 (92.90) |
| Uric acid (mg/dl) | | | 11–106 (36.50) |
| Osmolality (mosmol/L) | 680–830 | | |
| Lactate (mmol/L) | 320–470 | | |
| Other | Calcium oxalate crystals noted | | |

**Table 29.2.** Blood Urea Nitrogen Values (ISIS 2005)

| Sera Value (Units) | Asian (Standard Deviation) | African (Standard Deviation) |
|---|---|---|
| BUN (mg/dl) | 4.64 (±1.43) | 12 (±3) |
| Creatinine (mg/dl) | 1.595 (±0.396) | 1.5 (±0.5) |
| Uric acid (mg/dl) | .20 (±.31) | 0.20 (±0.4) |

## DISEASES AND DISORDERS

### Urolithiasis

In an 8-year-old African elephant diagnosed with chronic renal failure, two calcium carbonate stones were noted in the left ureter on necropsy. It was presumed that they were of some significance because both the proximal ureter and renal pelvis were dilated.[9]

In Germany, diagnosis of a larger stone in the urinary bladder via urethral endoscopy was noted; however, the consistency of the stone was not noted.[14]

### Chronic Interstitial Nephritis

Chronic interstitial nephritis and nephrocalcinosis has been reported in an 8-year-old female African elephant.[9] Clinical and laboratory signs paralleled those of renal failure in other species, e.g., depression, weight loss, and isosthenuria. Sodium, potassium, and chloride clearances were elevated when compared with three apparently normal elephants. Premortem diagnosis was made by paralumbar renal biopsy and presumptive diagnosis from urinanalysis. At necropsy, two calcium carbonate stones were noted in the left ureter and culture yielded a pure growth of *Pseudomonas aeruginosa*.

### Pyelonephritis

Two cases of pyelonephritis in Asian elephants have been noted—one by Sanchez[15] and the other by the author. In the first case, a 37-year-old female was presented for listlessness, dark-colored urine, and anorexia. A urinalysis showed hematuria, the presence of leukocytes, isothenuria, proteinuria, granular casts, and an absence of calcium oxalate crystals. There was a mild leukocytosis in the hemogram that increased to 32,200/ml on day 10, and there was also an increase in the serum creatinine and BUN. Culture revealed a pure growth, a *Streptococcus zooepidemicas* that was sensitive to cephalosporins, so the animal was started on 6 g ceftiofur IV TID. Treatment also included fluid supplementation via intravenous and rectal routes. Although the elephant appeared to make a full recovery, a post-treatment rectal ultrasound examination revealed moderate diffuse hyperechogenecity that in horses and other domestic species often correlates with tubular degeneration and subsequent fibrosis.[15]

The second case occurred at a Midwest zoo in a 32-year-old female Asian elephant. Her initial presentation was for a right rear leg lameness; however, as part of the examination, it was noted that her BUN was 47 mg/dl and her creatinine was 3.5 mg/dl. The WBC was within normal range (17,000 WBC/ml), but with a neutrophilic shift (13,400/ml), and a mild anemia (PCV = 28%) was also noted. It was suggested that the animal's water intake be monitored and that she be retested in 1 week. At that time, the WBC had risen to 21,300/ml with a heterophilia (9580 neutrophils/ml); the BUN was 41 mg/dl and the creatinine was 3.8mg/dl. A urinalysis was obtained 1 week later. In the interim, the elephant was started on 60 grams PO SID of a sulfadiazine trimethoprim combination. The subsequent urine sample that was obtained "free catch" showed 3–5 WBC/hpf, 1–3 RBC/hpf, SG = 1.009, and a pH of 6. The white count was further elevated (21,900 WBC/ml), including the appearance of 4% bands. Throughout this period her serum calcium and phosphorous levels were elevated (ranging from 13.7–15.2 mg/dl and 7.6–9.2 mg/dl, respectively). Antibiotic therapy was changed to 4.93 grams of enrofloxacin PO SID. Culture later yielded moderate growth of a *Corynebacteria* sp.; however, it is often a contaminant, and its significance was not certain. Due to increasing failure to accept the enroflox-

acin, antibiotic therapy was changed to 24 grams of ampicillin PO BID. The WBC during this period ranged from normal (13,500), with a continuing neutrophilia, to elevated as noted above. The animal appeared to improve clinically; however, 1 month after diagnosis, she was found dead in her stall. Postmortem examination revealed a yellow purulent material in both kidneys and the urinary bladder. Culture of the renal pelvis yielded *E. coli* (which had not been isolated from any of the urine cultures). There was also serosanguinous fluid in the vagina that yielded *Klebsiella pneumoniae* and *Enterobacter* sp. Histology revealed lymphoplasmocytic infiltrates and fibrosis suggesting chronicity in both kidneys. Mineralization of vessels, gastric glands, and lung tissue was further evidence of chronicity.

### Chronic Cystitis Associated with a Perineal Hernia

The author diagnosed a case of chronic cystitis in a female elephant whose bladder was entrapped in a perineal hernia. The hernia resulted from an unusually short labor (45 minutes) for a primiparous elephant. The presence of the bladder in the hernia was confirmed by ultrasound examination, and the entrapment resulted in only partial emptying of the bladder's contents. It was believed that the chronic urine stasis predisposed to cystitis. Treatment consisted of long-term antibiotics. Surgery was considered; however, after consultation with several equine surgeons experienced in elephant surgery, it was decided that the risk of surgery outweighed the risk of the ongoing cystitis.

### Follicular Vulvitis and Vaginitis

Follicular vulvitis and vaginitis was seen in a series of captive Asian elephants (62% of 29 sampled) and captive (89% of 19) and wild (90% of 30) African elephants.[11] Lesions consisted of hyperemic nodules ranging from 2–10 mm in diameter that would sometimes coalesce into plaques ranging from 8–10 cm in size. Histology of the affected tissues revealed reactive lymphoid follicles, but no specific bacteria nor viruses were associated with them (including specific attempts to isolate *Mycoplasma* and *Ureoplasma spp*.). The lesions were not associated with clinical signs in the captive elephants, and they were also found in 10/11 pregnant wild African elephants with no apparent effect, so they were considered to be of no clinical significance.[11]

### Polyps in the Urogenital Canal

Sizable polyps (up to 3.2 kg in one case) have been noted in the urogenital tract of female African elephants.[10] Histologically, the tissues were similar to fibropapillomas with cystic subsections that resembled Bartholin's glands. However, neither attempts at identifying papilloma virus by DNA probes (using bovine papilloma virus) nor with culture were successful in identifying a viral agent.

### Toxicities

Clinical trials with the aminoglycoside antibiotic, amikacin, at the rate of 6–8 mg/kg SID noted a mild reversible tubular insult in one of the five elephants treated.[5] The condition was characterized by a transient increase in creatinine and tubular casts in the urine. The authors presumed that, as in man, the condition regresses over a 20–60 day period, and if caution is used, does not preclude the use of amikacin in this species.[5]

### Other Diseases

Other diseases that cause vascular compromise may exhibit urinary tract manifestations among their many other systemic effects—petechial bladder hemorrhages, for example, have been reported in an elephant with anthrax and cowdriosis.[12]

One reference was made to the occurrence of cystic kidneys in elephants, with no further information given.[19]

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# 30 Nervous System

Michele Miller

## UNIQUE ANATOMY

The elephant's nervous system follows the same basic mammalian divisions.

### Central Nervous System

Elephants have the largest brains of any of the land mammals with reported weights of 3.6–6.5 kg, and those from African elephants are slightly larger and heavier.[2,10] When viewed from the dorsal aspect, the brain appears to be compressed at the cranial extent, widening at the temporal lobes. On the lateral view, the brain seems flattened, rather than rounded. The cerebrum has large frontal and temporal lobes but lacks an occipital lobe.[6] The large temporal lobes are part of the limbic system and in humans, associated with memory centers and learning.[13] The olfactory lobes are large and may compensate for the poorly developed visual system.[9] The hypophysis lies as a flattened tissue beneath the midbrain because there is no sella turcica in the sphenoid bone.[2]

The elephant cerebrum and cerebellum have a highly convoluted cortex. Studies of the fetal elephant brain indicate that the cortical folding changes continue to develop after birth, and it has been hypothesized that this may play a role in the long maternal dependence.

The spinal cord of the Asian elephant has been measured at 5.8 m in length.[9] It starts at the foramen magnum and ends at the level of the first sacral vertebra.[2] Although the cervical enlargement of the cord is small (C6–T1), the lumbar enlargement is large (L1–4).[9]

### Peripheral Nervous System

There are 12 pairs of cranial nerves and 40 pairs of spinal nerves in the Asian elephant.[9] The trigeminal nerve (V) is a large nerve that contributes a branch to form the proboscideal nerve. A branch of the facial nerve (VII) also contributes to the proboscideal nerve. The longest cranial nerve is the vagus nerve (X), which contains parasympathetic fibers. Branches of C6–8, T1 form the brachial plexus, and the lumbosacral plexus contains nerve branches from T19, L1–4, and S1–4. The innervation to the extremities is similar to that of domestic ungulates except for the forelimb digits.[9]

### Autonomic Nervous System

The autonomic nervous system is similar to that of domestic ungulates.[9] In elephants it plays an important role in thermoregulation.

## UNIQUE PHYSIOLOGY (SLEEP)

Both African and Asian elephants are diurnal. Although elephants can sleep standing, most studies indicate that animals sleep in lateral recumbency, if undisturbed.[14,15] Similar sleep patterns have been observed in free-ranging African and captive African and Asian elephants, with sleep periods lasting 3.1–6.9 hours, 1–4.5 hours in recumbency between 11 p.m. and 7 a.m.[8,14,15] During the night, animals will often get up to feed and then lay back down to sleep again.

## EXAMINATION OF THE NERVOUS SYSTEM

The neurological exam of the elephant should follow a pattern similar to that of domestic large animals.[3] The clinician may perform evaluation of the animal's sensory perception and motor responses during the physical exam (see Chapter 7). Other reflex testing is limited, but anal tone and cutaneous sensory response may be assessed subjectively.

Electromyography (EMG) is a diagnostic technique used to evaluate the electrical activity of a muscle by inserting a recording electrode. It is used to determine whether the site of lesion is in the muscle fibers or nerve. Although this technique has been anecdotally used to assess trunk paresis in elephants, no normal values currently exist for comparison.

Thermography has also been recently used in veterinary and human medicine for detection of abnormalities in the vascular, neural, and muscular systems, based on differential skin temperatures. This is a noninvasive method that uses a real-time infrared camera to visualize surface temperatures of the affected area. Although not diagnostic for a specific injury or condition, it has been useful in elephants for localizing areas of inflammation or injury, such as peripheral nerve disease.[5]

## DISEASES AND DISORDERS OF THE NERVOUS SYSTEM

### Infectious Diseases

Infectious diseases of the elephant nervous system are discussed in detail in Chapter 11. The important diseases that have been reported in elephants include rabies, encephalomyocarditis, and tetanus. Botulism and West Nile virus encephalitis are other potential diseases of concern.

A single case of fungal infection of the brain has been reported in a 6-year-old African female elephant that died of salmonellosis.[10] A granulomatous lesion was discovered at necropsy in the temporal lobe.

**Parasitic diseases.** Parasitic diseases that directly affect the nervous system have not been reported in the elephant.

### Noninfectious Diseases

#### Heat exhaustion/heat stroke.

Description. Heat exhaustion is an exposure syndrome characterized by rising body temperature. Heat stroke is the progression from heat exhaustion. See Chapter 17 for more details.

Predisposing factors. The usual cause is overexertion in hot weather. Obesity, humid conditions, inability to access shade or water, or debility may also predispose to overheating. Nervous or excitable elephants may also be predisposed.

Clinical signs. Clinical signs are fatigue or weakness, rapid breathing, ear droop, and flaccid trunk. The animal may tremble, become ataxic, and show signs of vascular collapse. Core body temperatures will be high. Eventually, an elephant will collapse, exhibit seizures, and progress to coma and death if left untreated.[4]

Diagnosis. Antemortem diagnosis results from evaluation of environmental conditions, rectal and skin temperature, and clinical signs. At necropsy, other diseases should be ruled out; the animal will probably show signs of terminal shock.

Differential diagnosis. Differential diagnoses include cardiovascular disease/failure, toxicities, EMC, acute septicemia, and other causes of shock.

Management. Immediately stop all activity. Give oral and/or rectal fluids. Intravenous fluids may be required in advanced cases. If sedation is required, avoid drugs that cause vasodilation or hypotension.

Prevention. Provide access to shade and water. Avoid working animals that are unfit; avoid working them during the hottest parts of the day or during adverse conditions.

#### Degenerative/inflammatory/idiopathic conditions of the CNS.

Description. This broad category encompasses conditions such as dementia, seizures, meningitis/encephalitis, and spinal cord neuropathy, which are fortunately rarely reported or observed in elephants.

Predisposing factors. Because the etiology of most of these conditions is unknown, the predisposing factors are also unknown. In one case, recurrent seizures in a rescued Asian elephant were related to a cranial injury to the occipital region that occurred at the age of 6 months, probably from a tiger attack (personal communication, Dr. Retno Sudarwati, Bogor, Indonesia, March 2005). See the Indonesia section in Chapter 35. Trauma, nutritional/metabolic imbalances, and exposure to toxic substances may also lead to clinical signs.

Clinical signs. Depending on the site and cause of the CNS dysfunction, elephants may be asymptomatic or display only mild vague signs. In other cases, such as in generalized meningitis/encephalitis, animals may show lethargy/depression, ataxia, abnormal mentation, apparent blindness, paradoxical excitement or agitation, seizures, coma, and death.[4] There are two reported cases of single seizures in adult African elephants.[10] One bull exhibited only muscle spasms and twitching; a cow displayed a more generalized seizure. No cause was found in either case.

Diagnosis. The diagnosis of seizures may be made on clinical signs, but antemortem diagnosis of meningitis/encephalitis is difficult in an adult elephant. Spinal fluid taps are not feasible antemortem, but fluid analysis, cytology, culture, and histopathological examination of tissues can provide a definitive diagnosis at necropsy. Careful evaluation to rule out trauma, exposure to toxins (through history, environmental assessment, serological, fecal, and feed screening), serial examination of the elephant's mentation, gait, and general physical state may provide clues to the underlying problem. CBC and serum biochemistry should be included in the diagnostic workup to rule out systemic problems, such as septicemia, hypocalcemia, hypoglycemia, or hepatic disease. Toxin screens may be run on tissue and GI contents postmortem.

One female Asian elephant with a sudden change in

behavior was found to have abnormal structures in her brain after euthanasia.[10] Spinal cords from a variety of aged mammals showed subclinical lumbar polyradiculopathy in nerve roots including an Asian elephant demonstrating degenerative changes in the myelin sheath.[1]

Differential diagnosis. Any debilitating disease may result in weakness, lethargy, and depressed mentation that may mimic a degenerative/inflammatory condition of the CNS. Behavioral changes may be a result of pathological disease or nonorganic causes. Degenerative/inflammatory CNS disease is primarily a "rule-out" diagnosis antemortem.

Management. Treatment of an elephant in seizure is dangerous and usually impractical. Administration of intravenous diazepam can be attempted, but because most seizures of an idiopathic nature are self-limiting, the most important actions are to prevent injury to the elephant and staff. If the underlying etiology can be determined, specific treatment should be targeted at the cause. Supportive care should be given in the form of decreasing stress, providing good nutrition, and controlling environmental factors.

Treatment of other neurological signs may include antiinflammatories or antibiotics, although these would be administered on a presumptive diagnosis (see Chapter 15).

**Trigeminal and facial nerve peripheral neuropathy.**

Description. The trigeminal (V) and facial (VII) nerves carry both sensory and motor fibers to the face and trunk.

Predisposing factors. Because the branches become relatively superficial on the side of the face and as they pass through various foramen and other structures, they are prone to trauma or disruption.

Clinical signs. Clinical signs depend on which branch is affected, but they may include lack of palpebral reflex on the affected side; eyelid ptosis; abnormal movement or floppy ear; and loss or abnormal sensation to affected areas of the mouth, trunk, or face, indicated by rubbing, abnormal mastication, or droopy lip and salivation. Damage to sensory fibers may be more difficult to detect than to motor nerves.

One young African elephant experienced a case of suspected trigeminal neuropathy after being laterally recumbent overnight during a case of colic. A week later he developed keratopathy due to inability to blink (personal communication, Dr. Pat Morris, San Diego, California, March 2005).

Diagnosis. Clinical signs lead to a presumptive diagnosis of trigeminal or facial neuropathy based on the knowledge of innervation to the head.[9] Symmetry of eyes, lips, trunk, and other muscles should be observed. Palpebral response and comparative evaluation of sensation on various areas of the face should also be assessed. Ability to prehend and chew food may yield important clues. History of anesthesia, prolonged recumbency for illness, trauma to the head, or other predisposing factors should be determined.

Differential diagnosis. Flaccid trunk syndrome, other cranial neuropathy, myopathy, cellulitis, and CNS disease should all be considered. Trauma may affect several cranial nerves simultaneously. Fracture of the zygomatic arch and trauma to the region over the eye left one female elephant blind after recovery (personal communication, Dr. Genevieve Dumonceaux, Tampa, Florida, March 2005).

Management. Supportive care including eye lubricants, hand feeding, dietary modifications to aid mastication, and treatment of any self-inflicted wounds are important to prevent secondary problems. If the neuropathy is caused by minor trauma, it may be temporary and resolve with time. Antiinflammatory drugs such as short courses of steroids or NSAIDS may decrease swelling around affected tissues. Local therapy to increase circulation may also aid recovery (e.g., hydrotherapy, application of DMSO).

**Radial nerve paresis/paralysis.** Although this syndrome is well described in large domestic animals, it has not been reported in elephants. Because the nerve is more protected by muscle and thick skin in elephants compared to horses, it seems less prone to injury.

**Trunk paresis/paralysis.**

Description. A wide spectrum of problems falls under this category and has been reported in both Asian and African elephants.[7,11] One specific syndrome, flaccid trunk paralysis, is described separately below. All other problems related to inability to use the trunk are included in this group, from mild weakness to complete paralysis and muscle wasting.

Predisposing factors. Proposed etiologies include trauma, infection, nutritional imbalances, toxins, and neoplasia.[11] In some cases, a known history of trauma or infection may precede the onset of trunk paresis/paralysis. However, in the majority of cases, the inciting cause is unknown. One female Asian elephant appeared to have temporary trunk paresis as an aberrant response to amikacin injections (personal communication, Dr. Genevieve Dumonceaux, Tampa, Florida, March 2005). There are also anecdotal reports of trunk paresis lasting 5–7 days after transport in older elephants (personal communication, Dr. Jackie Gai, Vacaville, California, March 2005).

Clinical signs. Signs may be mild and temporary, with only weakness or difficulty raising the trunk to mouth or prehending food. In other cases, paresis may be permanent and result in muscle atrophy.[5] Unlike the African elephant syndrome, the trunk paralysis cases in Asian elephants appear to happen slowly over a number of years. Affected elephants may learn to adapt by swinging the trunk to reach the mouth, using the feet or other objects to lift items, and leaning over to drink.

Diagnosis. Clinical signs of inability to use the trunk are usually apparent. Determination of the underlying cause may be difficult, especially if it is a chronic condition. Careful history-taking to rule out any potential traumatic incidents is critical. Feed analysis, CBC, serum chemistry and mineral panels, and toxin screen may also be helpful in identifying other causes. Thermography has been used to compare thermal signatures of affected (Fig. 30.1A, Color Section) and nonaffected (Fig. 30.1B, Color Section) elephants.[5] This may help outline impacted areas and aid in identifying potentially affected nerve tracts.

Another potential diagnostic technique that can be employed is EMG, although normal values have not been established for elephants (personal communication, Dr. Ray Ball, Tampa, Florida, March 2005). No histopathological findings have been linked to this syndrome to date.

Differential diagnosis. Diagnosis is based on clinical signs, although etiology may be difficult to elucidate.

Management. Treatment of specific etiologies, if determined, is recommended. However, in most cases, careful attention is needed to ensure that adequate nutrition and hydration needs are met if severe impairments are present. Administration of antiinflammatory drugs may be beneficial in acute onset cases.

There has been one reported treatment of trunk paralysis in an Asian elephant using acupuncture.[12]

### Flaccid trunk paralysis.

Description. Flaccid trunk paralysis is a unique syndrome affecting free-ranging African elephants in Lake Kariba, Zimbabwe and Kruger National Park, South Africa.[7] This problem was initially described as an outbreak in 1989 in Zimbabwe, with more than 30 elephants affected by an ascending flaccid paralysis of the trunk. At least 8 cases have occurred in one region of Kruger National Park.

Predisposing factors. The cases occurred in clusters at specific locations. Twelve cases were seen in 1989 near Fothergill Island in Zimbabwe. Additional cases have occurred since the initial outbreak when drought conditions had allowed access to grasslands adjacent to the island and mature bulls would take advantage to feed in this area during the rainy seasons.[7]

Clinical signs. Signs were observed in bulls 10–40 years of age. There was progressive loss of trunk function with ascending flaccid paralysis and muscle atrophy of the distal trunk muscles. Because the changes occurred over the course of several months, some of the animals learned compensatory methods to eat and drink. Despite this adaptation, some affected elephants became emaciated and disappeared. No obvious behavioral or other neuromuscular deficits were observed. None of the affected elephants have recovered use of the trunk.

Diagnosis. Because all affected elephants have been free ranging, diagnosis has been made on clinical signs. Histopathological samples from affected animals showed neuropathy of peripheral nerves supplying the trunk, with axon and myelin degeneration, without inflammation, and muscle atrophy.[7] There was no evidence of CNS involvement.

Differential diagnosis. Possible etiologies include trauma, infectious diseases, genetic predisposition, intoxications, and nutritional deficiencies. The most likely explanation based on the seasonal nature of the event and environmental assessment is plant intoxication.

Management. In severe cases, wildlife managers have culled affected elephants for humane reasons.

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# 31 Special Senses

Wm. Kirk Suedmeyer

## THE EYE

### Anatomy and Physiology

The elephant eye is comparatively small in relation to body size and is smaller than the whale, the elephant seal (*Mirunga* sp.), and the horse.[4,35] (personal communication, Dr. Gia Klauss, Columbia, Missouri, April 2005). It is generally similar in structure to other placental mammals. The average axial length of the African elephant eye is 35 mm,[4] and the average axial length of the Asian elephant eye is 38.75 mm.[10,23] The eye is located at a point halfway between the trunk and the auricular orifice.[30]

The pupil is circular (Fig. 31.1), and in one study of anesthetized African elephants (during daylight) the pupil measured an average of 2.5–4.0 mm in diameter.[35] The corneo-scleral envelope is unusually thick, particularly posteriorly, and devoid of any skeletal support structures (Fig. 31.2). The cornea consists of epithelium, Bowman's membrane, substantia propria, descemet's membrane, and endothelium. Evidence of Bowman's membrane in elephants is unusual in that this structure is lacking in the majority of mammals studied.[23] The iridocorneal angle is typical of large land herbivores, but it has a relatively underdeveloped aqueous outflow system.[5]

Elephants have a *Tapetum lucidum fibrosum*, a primitive reflective structure that develops from a thin layer of collagen fibers associated with the inner layer of small vessels of the choroids.[2,24] The dense fibers are closely woven together much like tendon sheaths, giving the tapetum its glistening appearance. The tapetum is dull yellow funduscopically and virtually identical in structure and function to other ungulates. To enhance vision in subdued light the tapetum serves to reflect light back onto the retina, increasing sensitivity to available light.[39] The tapetum has a rounded apex and straight base[32] and in general has a triangular shape, similar to felids. In addition, the border of the tapetum courses closer to the temporal than to the nasal margin of the fundus, as it does in felids.

The retinal vasculature is classified as paurangiotic—i.e., the vessels are minute and extend a short distance from the optic disc. Similar arrangements may be found in the Hyrocoidea, Sirenia, Guinea pig (*Cavia porcinus*), and other Perissodactyla. The paurangiotic retinal vessels of the domestic horse *Equus caballus*, tapir *Tapiris* sp., and rhinoceros *Diceros* sp. are present only at the optic disc. Optic disc size has been investigated during field immobilizations of African elephants. All adult elephants examined had optic disc measurements of 5 mm in diameter.[35] Research into the ganglion cell layer of the retina in African elephants has identified two distinct areas of increased ganglion cell density arranged along a horizontal axis extending across the retina inferior to the optic disc. This visual streak is comparable to other mammalian species, but a second retinal ganglion cell–rich area was also identified in the superior temporal retina in a pattern that is distinctive for elephants. It is postulated this is an adaptation to monitor the movement and appearance of the trunk.[32] During this same investigation, researchers found no evidence of an *area centralis*, or cone-rich area.[32]

Documentation of visual acuity has been based more on ophthalmic anatomy than ocular reaction and sensation. A correlation exists between the maximum density of ganglion cells (in the elephant 550–610/mm$^2$) and the size of the eye, which in one investigation of a juvenile African elephant determined a value of 4.05 cycles/degree.[32] This acuity is less than domestic cats, but greater than domestic rabbits. The authors caution that a mature elephant may have greater acuity owing to the progressive growth of the eye as the elephant matures.

The perception of color vision in elephants is unknown, with arguments for and against.[30] During a preliminary study of memory in an Asian elephant, the animal made fewer mistakes with green and blue blocks than with red, yellow, or white.[19] However, identification of cones, color sensitive receptors of the retina in other species, has not been documented in elephants.[23]

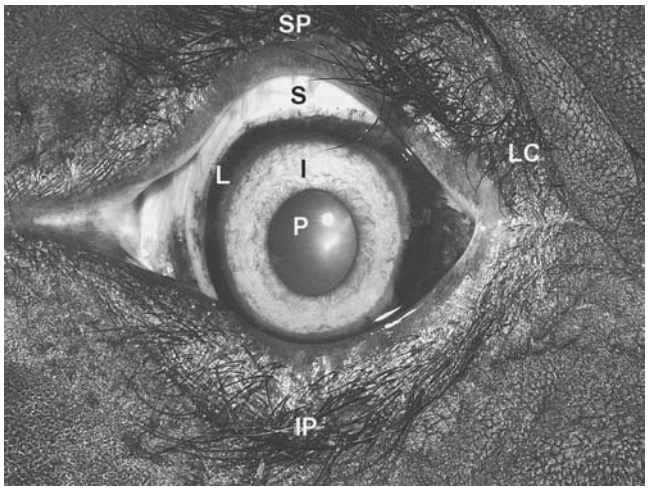


***Figure 31.1.*** *Asian elephant eye (O.S.) demonstrating salient clinical features. Medial canthus, lateral canthus (LC), superior palpebrae (SP), inferior palpebrae (IP), limbus (L), sclera (S), pupil (P), and iris (I). (Photo courtesy of Dr. Pearce, UMC College of Veterinary Medicine, Columbia, Missouri.)*

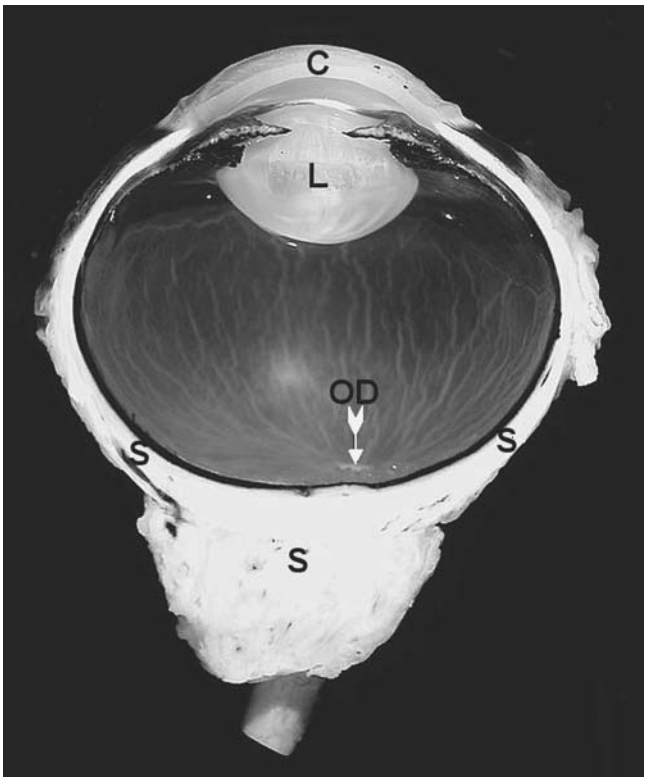


***Figure 31.2.*** *Saggital gross section of an Asian elephant eye. Note the cornea (C), lens (L), optic disc (OD), and sclera (S), especially the extremely thick postretinal sclera. (Photo courtesy of Dr. Dubielzig, University of Wisconsin-Madison School of Veterinary Medicine.)*

The iris varies in color from tan, yellow, brown, or green to light blue[30,35] or combinations thereof, and may contain "iris freckles," a collection of chromatophores (see Figs. 31.1 and 31.3). In contrast to horses, *corpora nigra* are absent.

The lens is crystalline, round, and smaller than other ungulate species such as kudus *Tragelaphus* sp., gemsbok *Oryx gazella,* and hartebeest *Alcelaphus caama*. This confirms Haller's ratio of an inverse relationship of eye size to body size.[35] The lens of the eye grows in a rectilinear fashion throughout the life of African and Asian elephants.[16] Initially, there is rapid growth followed by slow progressive growth. Lens weight can be used to estimate the age of elephants postmortem, especially after 8 years of age,[16] though the author cautions that captive animals appear to grow more rapidly than their wild counterparts.

The ocular adnexa of elephants parallel most other placental mammals. Accumulations of brown to black pigment measuring 4–10 mm involve the limbus and conjunctiva, may extend onto the cornea (see Fig. 31.1 and 31.3) and are contained within branching contractile cells. A white, circumferential ring, similar to the *arcus seniles* in man is also present in both species and may be associated with age.[28,35] See Figure 31.3.

Conjunctival tissue appears to have similar structure and function as in domestic animals. Three eyelids are present in the elephant: superior and inferior palpebrae and a nictitating membrane. Eyebrows are absent in elephants.[39] The lower eyelid is more developed and ascends to a greater degree than the upper lid descends.[4,39] The upper lid contains a supportive tarsal plate composed of dense fibrous tissue.[30] Although most mammals have true eyelashes, elephants do not.[39] Large (up to 110 mm), superior periocular lashes extend over the eye, effectively shading and protecting the eye.[35] The superior lid contains the *levator palpebrae superioris* muscle that functions to elevate the superior lid. The inferior lid also contains periocular lashes, but they are finer and extend over a shorter margin than the superior lashes.[35] These lashes seldom exceed 20 mm in length.[34] The lower lid is moved ventrally by the *depressor palpebrae inferioris* muscle.[18] Both lids close via the annular orbicularis oculi muscle.

The nictitating membrane contains a small but well-developed harderian gland that appears to function in a similar fashion to other mammalian species. It opens on the medial aspect of the nictitating membrane and is the main source of lubrication for the corneal surface. The nictitating membrane of elephants also contains a branch of the *annular orbicularis oculi* muscle, which controls movement over the eye.[30] The Meibomian (or tarsal) glands, found in most mammals, are replaced by Zeis's glands in the elephant and are located in the lid margin. Zeis's glands, modified sebaceous glands, produce an oily secretion that helps lubricate the cornea.

The angle between the optic axes and midline is 55° and is identical for the elephant, cavy, deer, ox, and sheep.[31] The binocular field for the elephant is 67°[4] comparable to the horse (60–70°). The uniocular field is 123°, compared to 146° for the horse.[4]

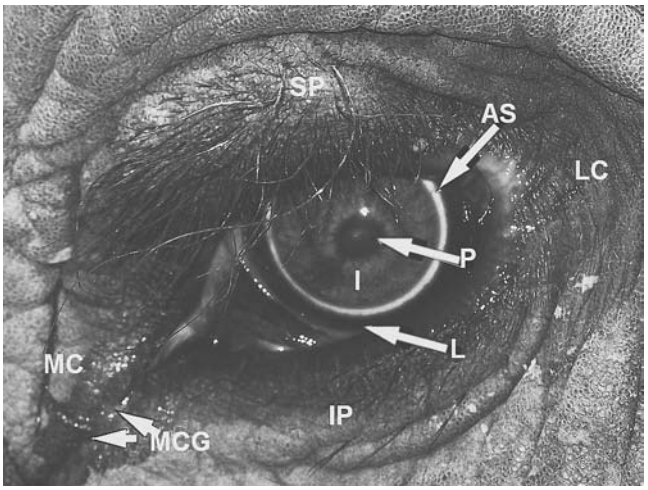


***Figure 31.3.*** *African elephant eye (O.S.) demonstrating salient clinical features. Medial canthus (MC), medial canthal groove (MCG), lateral canthus (LC), superior palpebrae (SP), inferior palpebrae (IP), iris (I),* Arcus senilis *(AS), pupil (P), and limbus (L). The iris is heterochromatic.*

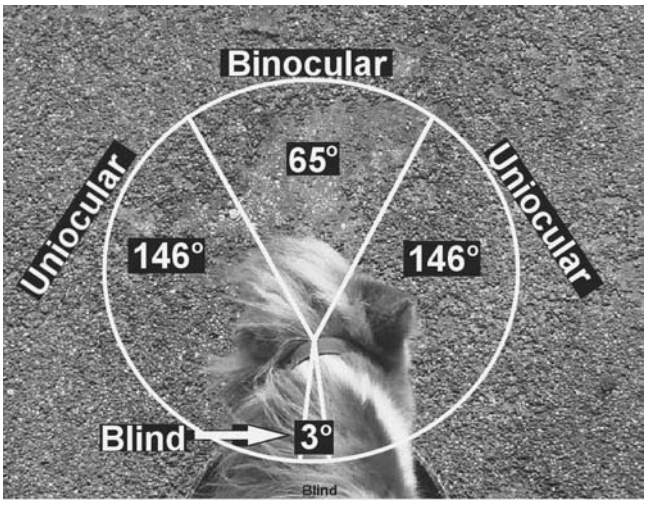


***Figure 31.4.*** *The total visual field for a domestic horse* (Equus cabellus). *The binocular overlap is 65° with a blind spot of 3°, allowing an equid to see a field of view of 357°. Compare this to the total visual field for an elephant (Figure 31.5).*

Thus, the monocular field for each eye (including binocular overlap) is 190°, compared to 211° in the horse (Figs. 31.4 and 31.5). The elephant's large head and laterally set eyes dictate a large interpupillary distance (730–1130 mm).[30,35] Elephants must converge their eyes and apparently raise their head slightly to focus with binocular vision.[35] This is apparent when observing elephants from a frontal view—both eyes are easily seen and thus the reverse is assumed to be true.[32]

A unique feature of the elephant eye is the lack of a lacrimal apparatus.[4,18,30] Tear films simply flow towards the medial canthus and exit along a groove in the skin onto the face in Asian elephants.[23] We have observed a similar groove (medial canthal groove) in African elephants (see Fig. 31.3). This should not be confused with epiphora or dacryocystitis associated with ocular abnormalities in other animals.

## Ophthalmic Examination

Ophthalmic examination of the elephant eye should be performed in conjunction with the annual examination. Most elephants in free contact may readily be examined. Constant and appropriate communication between the handler and the clinician is critical to safety. Protected contact animals may be more difficult to examine fully without stationary conditioning, sedation, or use of an elephant restraint device.

In most instances, the eye may be examined with standard ophthalmic techniques, including direct ophthalmoscopy, binocular and monocular indirect ophthalmoscopy, and retroillumination, though few reports of any systematic ophthalmic exams exist, and none are reported in awake elephants. Gonioscopy, ultrasound, tonometry, and slit-lamp examination has not been reported in elephants. During etorphine hydrochloride[a] immobilization, the eye remains open and the pupil is miotic and centrally placed, allowing ophthalmic examination, though pupillary light response is negated.[35]

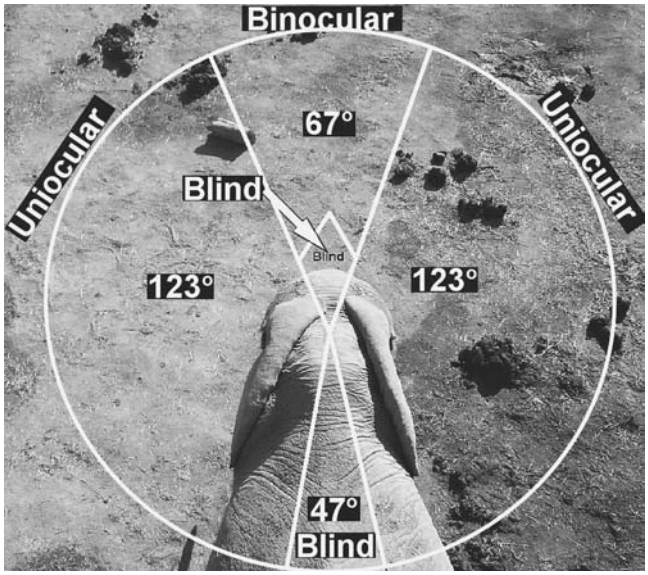


***Figure 31.5.*** *The total visual field for an elephant. Note the binocular overlap of 67° with a blind spot of 47,° allowing an elephant to see a field of view of 313°. There is a small blind spot just in front of the trunk.*

A visual examination is conducted first; use of additional illumination with a penlight or small flashlight may enhance the examination owing to the significant shadowing effects of the bony orbit, skin color, integumentary fissures, and superior periocular lashes. The in-

ferior palpebra is moist, and at times significant dampness may be visualized due to the lack of nasolacrimal passages.

Schirmer tear tests have been evaluated in clinically normal Asian elephants[36] without topical anesthetic agents. A mean value of 34.3 + 1.7 mm/min was determined for 80 elephants. Tear production differed according to age, with older elephants (41–60 years old) having higher values than younger (0–20 years old) elephants.

The eye and adnexa are examined and compared to the contralateral eye for symmetry and conformation from several angles (i.e., frontal, lateral). Nictitating gland position; globe size, position, and appearance; iris color; and pupil symmetry should be evaluated. The latter can be performed from several feet away in the subdued light of an indoor stall. Anisocoria, though not reported in elephants, should be detected with retroillumination from tapetal reflection. Ocular discharge, erythema, and edema should be noted and examined further.

The cornea should be grossly examined for clarity and surface irregularities. Elephants routinely demonstrate astigmatism that is thought to have corneal and lenticular components.[23] Gross opacities of the anterior chamber and lens may be observed through retroillumination with light reflected by the tapetum.[20]

Conjunctival cultures may be performed as they are in domestic animals; however, the strength of the palpebrae may overcome attempts at culture and may necessitate use of topical anesthetics. In a study of 44 normal elephants, 63 of 79 conjunctival swabs were positive for aerobic bacteria or yeasts. Gram-positive organisms (primarily *Staphylococcus* spp. and *Corynebacterium* spp.) were identified in over 50% of the isolates. *Acinetobacter lwoffii* was the main gram-negative bacterium identified.[37] No untoward effects from standard ophthalmic preparations have been reported in elephants. The author has cultured elephant conjunctivas without the need of topical anesthetics. The clinician is cautioned that topical anesthetics are commonly bactericidal and it is suggested that if topical anesthetics are employed, the contralateral eye should be cultured for comparison.[20]

Pupillary light response (PLR) is easily evaluated if the clinician can safely approach the eye. Use of an appropriate illumination device (Finoff transilluminator) in subdued light elicits the best response. Consensual response is difficult to assess due to the significant interpupillary distance. In theory, because elephants lack an *area centralis*, a more complete PLR may be elicited when light is focused in the temporal or nasal retina; ganglion cells are concentrated along the visual streak (as in other ungulates) and the upper temporal area of the retina, though this has yet to be evaluated in elephants.[32]

Use of fluorescein stain[b] may be difficult to perform in the elephant due to the close proximity with which the operator must complete the procedure; blepharospasm might incur when attempting application. Diluting a fluorescein strip in sterile water and utilizing a standard syringe with a blunt small gauge needle or IV catheter allows the operator to spray the stain across the eye, either from the medial or lateral canthus. Applying a steady stream of water directed at the periocular skin will usually cause the elephant to relax and allow application of the ocular stain. Direct application is nearly impossible due to the efficiency with which the superior periocular lashes protect the eye.

Flushing the eye removes excess stain. As with domestic animals, loss of corneal integrity allows uptake of fluorescein. Use of a small portable black light in low-light conditions illuminates the fluorescein, detecting corneal damage. Because elephants lack a nasolacrimal apparatus, fluorescein will not be observed in the oral cavity or the distal end of the trunk, but will overflow the inferior palpebrae via the medial canthal groove.[18,30]

Direct ophthalmoscopy remains the standard method for examining the eye. Evaluation should proceed with visualization of the optic disc, paurangiotic retina, tapetum, lens, iris, anterior chamber, and cornea. Examination of the iridocorneal angle is best achieved through gonioscopy and has not been documented in awake elephants. Colobomas and glaucomatous cupping, though not reported in elephants, are observed at the optic disc in domestic animals.[20] Optic discs appear creamy white to off-white in color in elephants. Diopter selection allows magnification and evaluation of various components of the eye.

Indirect ophthalmoscopy, utilizing either binocular or monocular vision, has not been reported in elephants. Video and "super" retinoscopy has been utilized in anesthetized and awake elephants to determine the ocular resting refractive state.[23,35]

Vision evaluation in elephants is extrapolated from and similar to techniques used with domestic animals, especially the horse. Because elephants appear to alternate monocular with binocular vision,[32] partial evaluation of adequate vision may be accomplished by observing prehension of a small piece of a favored food item placed to the lateral aspect of the head. Alternatively, large cotton balls may also be used.

The menace response is an easily performed action toward the eye that elicits a blink response. In the African elephant, the author has easily elicited an appropriate response by simply moving fingers toward the eye, being careful not to create air movement that may elicit a tactile response. The menace response should, in combination with PLR and ERG, help the clinician locate visual reflex lesions, though once again, reports of vision evaluation in elephants are lacking.

To determine visual response in light of clinical cataracts, and/or iris atrophy and synechia, a clinician should also be able to perform the "dazzle reflex."[20] A very bright light is directed upon the visual streak of the

retina, eliciting bilateral narrowing of the palpebral fissure. A positive response indicates that the lower visual system (i.e., retina, optic nerve, chiasm, optic tract, and supraoptic nuclei) is intact.

### Diseases/Disorders

**Conjunctivitis.** Conjunctivitis was noted in 38 events involving 108 elephants during a review of disorders of the eye. Bulbar conjunctivitis was noted in most of the elephants undergoing optometric examination during field immobilizations of 21 African elephants.[35] Nodules and phlyctens (small lymph-containing vesicles) were often visualized, similar to phlyctenular conjunctivitis in people.[35] Similar lesions were noted in 126 of 300 African elephants culled in Tsvao National Park in 1966.[21]

Histologic examination demonstrated dense foci of lymphoid cells and prominent reactive centers and occasionally invaded the squamous epithelium of the conjunctiva. These lesions were similar to those found in humans affected with chronic irritation or viral infection, although the palpebral conjunctiva is most often affected in man. Lesions in these elephants were often restricted to one eye, though specific numbers were not reported.

Conjunctivitis has also been associated with poxvirus infection, especially in Asian elephants.[13] This disease is discussed in detail in the Chapter 11. Treatment of conjunctivitis depends upon the etiology and proceeds in accordance with standard ophthalmic techniques, taking into account the unique aspects of ocular anatomy and physiology.

**Corneal lesions.** Corneal lesions are occasionally reported in elephants. Traumatically induced corneal opacities were commonly observed in one field study of eye lesions in African elephants, though specific numbers were not provided.[21]

A persistent corneal erosion was treated in a 19-year-old Asian elephant. The elephant presented with blepharospasm and excessive lacrimation. Initial treatment consisted of topical chloramphenicol and dexamethasone drops. Upon examination, central corneal edema, a roughened epithelial surface, multiple white foci in the anterior stroma and miosis were observed.[41] The elephant was subsequently treated with topical atropine sulfate and chloramphenicol. No improvement was noted and the elephant was sedated to facilitate fluorescein staining. Corneal uptake was noted over a 7 mm diameter area of the cornea. The corneal defect was debrided and a striated keratotomy was performed in conjunction with lateral tarsorrhaphy and placement of an equine contact lens. The corneal defect healed without additional therapy.

A corneal foreign body, in the form of a plant thorn, was surgically removed under ketamine hydrochloride and acepromazine in an 11-year-old Asian elephant.[1]

Hypopyon keratitis was reported in the right eye of an Asian bull elephant.[33] The elephant presented with profuse discharge and "impaired vision." Examination revealed an accumulation of purulent debris within the anterior chamber, ulceration of the cornea, and keratitis. The eye was initially treated with a combination of zinc sulfate, boric acid collyrium, and topical chloromycetin. Atropine sulfate 1% was applied topically to prevent synechia formation. Placental extracts and cephaloridine were administered subconjunctivally. Hydrocortisone, vitamin A, and tetracycline hydrochloride were used as adjunct therapies and the lesion fully resolved.

Panophthalmitis was documented in nine eyes postmortem during a field study of eye lesions in African elephants.[21] Keratitis, iridocyclitis and retinitis were present. No signs of active or acute infection were observed and all lesions appeared chronic. Diagnosis of corneal disease is based on presenting signs (blepharospasm, epiphora) and results of ocular staining. Treatment depends upon ocular findings and follows generally accepted standards in small and large animal ophthalmology.

Subdermal injection of placental extract (Placentrex®)[g] is a common treatment for corneal opacities in elephants in Asia[40] (also personal communication, Dr. Khyne U Mar, United Kingdom, March 2005). See Figure 35.4 in Chapter 35.

**Lens.** Cataracts, lens luxations, and lens dissolution have been documented on occasion in both species.[21,38] Sixteen of 300 elephants demonstrated cataract formation, lens dissolution, or luxation in a field study of eye lesions in African elephants.[21] The authors speculate that lesions of the lens and retina may be the result of excess infrared and visible radiation due to limited shade and reflection of heat from the ground. During a review of disorders of the eye, 5 of 108 animals were diagnosed with cataracts. Two of these elephants had bilateral cataract formation. Cataract removal with phaecoemulsification has been reported in one Asian elephant, though specific details were not provided.[38] Total lendectomy of one eye was successfully performed to correct blindness due to a mature cataract.[15]

One enucleation in an African bull elephant was reported during a review of disorders of the eye. Facial nerve paralysis was thought to have caused the eye to become dry and infected; additional details were not provided.[22]

Standardization of ophthalmic techniques, results of ophthalmic exam, and documentation of ophthalmic disease is necessary to provide a full understanding of ophthalmology in elephants.

## THE EAR

### Anatomy and Physiology

The ear of the African elephant is large and shaped much like the continent of Africa. The pinnae extend above the neck, whereas in the Asian elephant, the ear is

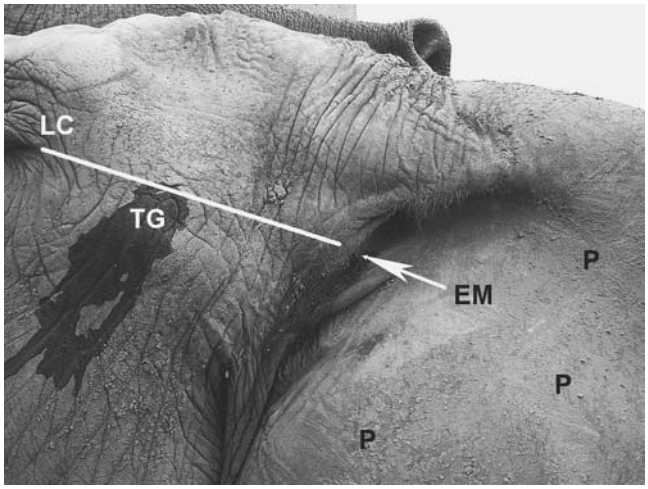


***Figure 31.6.*** *The left lateral aspect of an African elephant head. The external auditory meatus lies along a tangent from the lateral canthus of the eye (LC), through the temporal gland (TG), to the external meatus (EM). The large surface of the pinna is denoted (P). Rostral and caudal orientation are noted.*

relatively small and trapezoid in shape and positioned below the neck. In both species, the ear is sparsely haired. The ear is supported by a lattice-like plate of cartilage that does not extend to the full margin of the pinna. An extensive network of arteries and veins is present on the caudal surface. These vessels are commonly utilized for phlebotomy. The external meatus or auricular opening lies in the same plane as a line drawn from the lateral canthus of the eye, through the temporal gland and to the external meatus (Fig. 31.6).

The external meatus is surrounded by hair upon a structure analogous to the tragus in humans. An auditory tube connects the middle ear with the pharynx. In the Asian elephant, the auditory tube is fibrocartilagenous proximal to the pharynx and osseous proximal to the middle ear.[18] There are several functions to the ear, including acoustics, balance, thermoregulation, and conspecific signaling. The ear is an integral part of thermoregulation in both species of elephant.[42] See the section titled "Thermoregulation" in Chapter 17 for more details.

Hearing in elephants is acute,[7,8] and may be augmented by the pneumatic bones of the skull, though the latter has yet to be proven.[30] Elephants position the ears to localize sound and alert or otherwise communicate with other elephants.[3,8,9,25] Elephants were the first terrestrial animals found to produce infrasonic sounds. It is postulated that use of infrasonic calls allows communication over great distances.[25] A unique aspect of hearing in elephants is their inability to hear above 10,500 Hz during an intensity of 60 dB (decibels). Even at higher decibels, the elephant appears incapable of hearing above 12,000 Hz.[6] As a comparison, humans can hear up to 19,000 Hz and domestic dogs 44,000 Hz. However, elephants can hear low-frequency (infrasonic) sounds better than any other animal tested.[6,25] It has been demonstrated that elephants can hear at frequencies below that of humans, to levels of 17 Hz compared to 29 Hz in humans. It is theorized that the reason elephants cannot hear ultrasonic or high-frequency sound is due to the functional interaural distance. In essence, the larger the interaural distance, the more the ability to hear high-frequency sound decreases.[6] Sound waves reach one ear sooner than the other due to this distance. In addition, localization of sound in animals relies on two principles: 1) the time of arrival to the ear, and 2) the intensity of the sound upon the ears. Because there is a large interaural distance in elephants, sound reaches one ear long before the other, allowing the nervous system to determine the source readily.[6] In addition, the intensity of sound on the ear closest to the sound is greater than the opposite ear, due to the buffering effects of the head. The opposite is true with animals having short interaural distances; there is essentially no buffering of sound by the head, and the intensity is not decreased, forcing the animal to be able to hear high-frequency sound to determine origin.[6] Research in this area is ongoing (see the section "Additional Readings," later in this chapter; also of interest is The Savanna Elephant Vocalization Project/Elephant Voices initiated in 2001–2002 by Joyce Poole and Petter Granli, http://www.elephantvoices.org.).

## Examination

The ears should be cleansed of dirt and debris before examination. Elephants that are conditioned to allow physical examination of the pinnae facilitate examination. Both ears should be examined for symmetry, conformation, lesions, mobility, and auricular discharge. Purulent discharge or malodor emanating from the meatus should be evaluated further through culture and cytology.

Inspection of the external meatus is best performed with a strong light source due to the relatively small opening. Specific lesions of the auditory canal have been unreported in elephants. Elephants appear to have the ability to close the external meatus through a combination of pinna positioning and apparent muscular contraction of the overlying tragus (personal observation), though anatomical documentation of this ability is not reported. Phlebotomy scars and perivascular fibrosis are commonly observed lesions in those elephants routinely bled from the ear.

Specific auditory evaluation is difficult to perform due to a lack of standardized testing and necessity of specialized equipment. Cursory evaluation may be assessed through response to vocal commands by the trainers and keepers, though visual recognition of movement may complicate interpretation.

## Diseases

Diseases of the elephant ear are rarely reported in the literature, though traumatically induced lesions are

common and readily apparent. An aural rhabdomyosarcoma was surgically removed from an African elephant under general anesthesia.[17] The tumor was located on the anterior aspect of the pinna and had slowly progressed in size over a period of 4 years. The mass extended ventrally to the zygomaticoauricularis muscle (which controls ear movement) but did not involve the auricular cartilage. Though mechanical dehiscence was noted, the wound healed by secondary intention.

Although it has been reported that the ear should demonstrate no discharge from the external meatus, an oily secretion in clinically normal African and Asian elephants has been reported.[29] Purulent discharge was noted in 10 of 35 occurrences of otic discharge involving 43 animals. Details of treatment were not reported. In this same survey, 60 events involving the ears in 43 elephants were reported, including purulent discharge, bacterial infection, parasites, trauma, edema, pain or discomfort, erythema, and hemorrhage.[22] The author has observed secretions in both clinically normal and ill African elephants. In the ill animal, *Candida* sp. was cultured. This organism was not found in clinically normal elephants.

**Malpositioning.** During a survey of auricular problems in elephants, 15 events were classified as malpositioning of the pinna. Observed conformity problems included flopped, crimped, bent, and "held out." The majority of cases were unilateral in nature. No specific etiologies or treatments were reported.

**Vascular damage.** Phlebitis, vasculitis, and thrombosis have been documented in elephants. Thrombosis of the right pinna in a female African elephant was observed 48 hours after intravenous injections of flunixine meglumine[c] and trimethoprim-sulfadiazine[d]. Circumferential necrosis of >75% of the pinna necessitated removal (Fig. 31.7).

In this elephant, a line of demarcation between healthy and necrotic tissue facilitated removal, which involved debridement and removal of cartilage below the level of viable skin margins. The resultant wound was treated topically with 0.3% hydroxyquinolone sulfate ointment[e] daily for 5 months and fully resolved. Loss of thermoregulation has not been documented in this animal and no apparent clinical effects have been observed.[34] A conspecific elephant received intravascular flunixine meglumine and gentocin sulfate[f] and demonstrated no untoward effects.

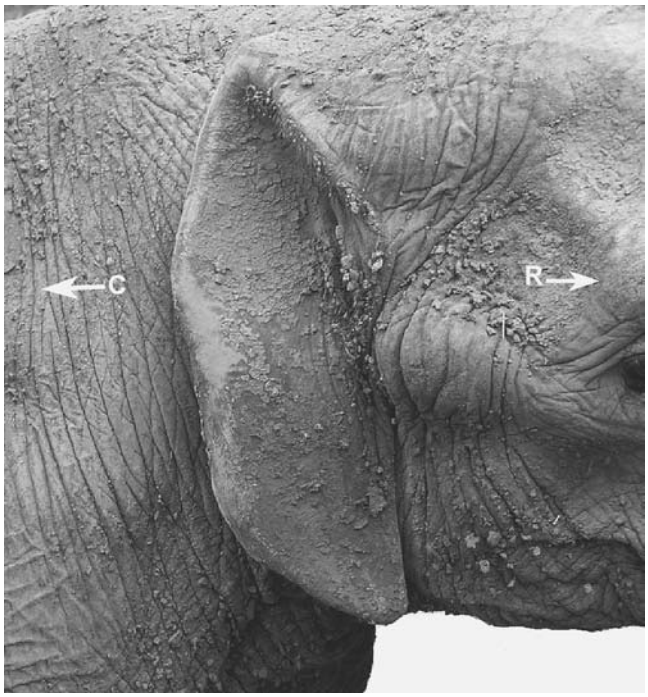


***Figure 31.7.*** *Postthromboembolic loss of 85% of the right ear of an African elephant. Rostral (R) and caudal (C) orientation are denoted. Necrosis of the pinna resulted from the administration of intravenous medication to treat a* Salmonella typhimurium *infection.*

## ACKNOWLEDGMENTS

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[a]Etorphine hydrochloride, Wildlife Pharmaceuticals, Fort Collins, Colorado 80522.
[b]Bio glo, Rose Stone Enterprises, Alta Loma, California 91701.
[c]Banamine, Schering-Plough Animal Health, Union, New Jersey 07083-1982.
[d]Tribrissen 48% Injection, Coopers Animal Health Inc., Mundelein, Illinois 60060.
[e]Bag Balm 0.3%, Dairy Association Co., Lyndonville, Vermont 05851.
[f]Gentocin sulfate injection, Vedco Inc., St. Joseph, Missouri 64504.
[g]Placentrax, Albert David, Kolkata, India.

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# 32 Chemical, Tactile, and Taste Sensory Systems

L.E.L. Rasmussen

## INTRODUCTION

The two largest land mammals, the Asian elephant, *Elephas maximus*, and the African elephant, *Loxodonta africana,* are heavily endowed with tactile senses and chemodetection systems. Especially well studied has been the reliance of the Asian species on chemical senses in many aspects of its complex lifestyle, including foraging, migration, reproduction, recognition of familial relationship, and maintenance of social structure.[35,37,39,40,42,43,45,58]

The deciphering of chemical signals and the discovery of two pheromones in this species has evolved into delineating how these signals are received and how they affect behavior of both wild and captive elephants, clearly demonstrating important social and reproductive functions.[25,26,49] Male Asian elephants track the proximity of females to ovulation by monitoring concentrations of female urinary (*Z*)-7-dodecenyl acetate (*Z*-7-12:Ac).[49] Females detect the correct ratio of ketones that signal musth or, remarkably, respond differentially dependent on their estrous phase to a bicyclic ketal (frontalin) released in the temporal gland secretions (TGS) of older adult males in musth; follicular phase females are attracted, luteal phase females are indifferent, and pregnant females avoid this pheromone.[48] Recent evidence suggests that the African species, perhaps with less emphasis, use chemical sensory information in their reproductive scenario (Schulte, unpublished). Almost all the information in this chapter relates to either the Asian elephant or the savannah species of African elephant because no detailed sensory information is available on the recently recognized forest species.

## TOUCH: ANATOMICAL AND FUNCTIONAL ASPECTS

### The Proboscis and Its Tip

**Muscular features.** Morphologically the trunk is a prehensile elongation of the upper lip and rhinarium (nose). The proboscis or trunk consists wholly of muscular and membranous tissue; the nasal cartilages serve only as valves at entrance to bony nares or turbinals.[28] The trunk is a tapering muscular structure of nearly circular cross-section extending proximally from attachment at the anterior nasal orifice, and ending distally in a tip or finger. The length may vary from 1.5–2 meters or longer depending on the species and age. The relaxed, fully extended trunk of an Asian female measured 1.5 m.[57] The trunk length of two adult African elephants, a 27-year-old female and a 22-year-old male, both measured 1.8 m, whereas that of male and female newborn African calves measured, respectively, 0.28 m and 0.38 m (personal communication, Heidi Riddle, Greenbrier Arkansas, May 2005). The Asian trunk has been reported to be more extensible.[52] The taper may change fourfold from the attachment at the skull to the finger; for example, in an Asian female the outside diameter at the head was 32 cm but only 8 cm at the tip.[57] In the Asian species external annulations are more regular and numerous.

Four basic muscle masses (the radial, the longitudinal, and two oblique layers) and the size and attachment points of the tendon masses allow the shortening, extension, bending, and twisting movements accounting for the ability to hold and manipulate loads up to 300 kg. The longitudinal muscles, subdivided into anterior, lateral, and posterior, are more superficial than the deeper radial and transverse muscles.[28] By employing these transverse, longitudinal, and oblique bands of muscles, elephant trunks, vertebrate tongues, octopus arms, and squid tentacles achieve exceptional flexibility and mechanical manipulation abilities. Octopuses recently have been demonstrated to have bipedal locomotion with support for this multiplex muscular action coming from a constant internal volume.[20] In the elephant trunk the added load-carrying advantage is explained by the tendon masses and their attachments.[57] Muscular and tendinous ability combined with nervous control allows extraordinary strength and agility move-

ments of the trunk, such as sucking and spraying of water or dust and directed air flow blowing. For example, elephants can triangulate and bounce food objects off walls by directing forceful air flows at the proper angle to repel such objects back to within their reach.

**Neurological features.** The trunk is innervated primarily by the maxillary division of the fifth cranial nerve (trigeminal), thus differing from other mammals whose external rhinarium are innervated by the ophthalmic branch. The maxillary branch emerges from the infraorbital foramen. At the root of the proboscis it divides into three branches: the dorsal branch supplies the lateral nasal region, the middle branch unites with a branch of the facial nerve forming the proboscideal nerve, and the ventral branch ramifies and supplies the ventrolateral portion of the trunk.[27] In other mammals, and presumably in elephants, the nasopalatine and ethmoid branches of the trigeminal nerve scatter finely myelinated A-delta and unmyelinated C fibers throughout the nasal epithelium that terminate in free nerve endings, functioning as peripheral chemoreceptors. In contrast, in the oral cavity the mandibular nerve carries sensory information from the mucous membranes of the mouth. In addition, trigeminal fibers bring somatosensory information (touch, temperature, pain) to the central nervous system.[4] For the elephant these free nerve endings of the trigeminal system provide the ability to detect irritating, noxious chemicals. Trigeminal threshold responses are generally at least a thousandfold higher than olfactory thresholds, although thresholds to acids may be as low as 10 ppm.[4] Irritants such as capsaicin in chili peppers forms the basis for one developed repellent system, although again requiring fairly high levels of the active substances.[31]

**Inner trunk features.** The interior of the trunk is lined with epithelium and many mucous glands and is densely innervated by free nerve endings. Glands near the base of the trunk produce extensive, acidic mucous secretions containing high concentrations of odorant binding proteins (OBPs). These lipocalin-like proteins play a significant role in olfaction, both as transporters and scavengers in the Asian elephant.[25] Specifically, excess *Z*-7-12:Ac pheromone, released from its urinary albumin transporter is removed by these OBPs.[25]

**Functions of the trunk.** The trunk is a multifunctional organ used for manipulating, lifting, eating, breathing, vocalizing, social disciplining, determining scent direction, and delivering volatile odorants to the main olfactory system and less volatile odorants to the vomeronasal organ (VNO) by a different adaptation.

Breathing function. The trunk is an essential part of an unusual respiratory design for a terrestrial mammal, perhaps indicative of elephants' postulated aquatic origin.[9,10,14,54,55] Through the lengthy trunk 70–80% of breathing occurs.[2] The truncal passageway effectively warms incoming air well in advance of reaching the trachea; it cools departing carbon dioxide–laden, expiring air.[47] The transit up the long trunk also warms and cleans that portion of the inhalant air that will pass through the turbinals.

Vocalizing function. Air blown out of the trunk is modulated by muscle compression, acoustically similar to wind instruments; examples include squeals of playing elephants, screams of angry elephants, and trumpeting. Annoyed elephants may repetitively thump their trunks on objects or the ground.

Social disciplining tool. The trunk is an effective disciplinary tool. Mother elephants restrain and forcibly move their calves out of danger, older females may swat younger, pestering males, and matriarchs may swing at unruly younger females.

Scent directionality determination. Experienced elephants use an uplifted trunk as an odor and wind periscope. The direction of an odor is determined by varying the periscope direction coordinated with sniffing to achieve inhalation of air sufficient to reach olfactory sensory areas.

Transporting odorants. The trunk warms odorants and transports them to the main olfactory systems. Neurosensory cells of nasal epithelium are protected by the extensive mucous system. In addition, the tip is intimately involved in ensuring that less volatile odorants and pheromones reach the ducts of the VNO.

**The tip.** The trunk tip or finger of the two species differs at the anatomical level. The African species has ventral and dorsal processes whereas the Asian trunk tapers to a single exaggerated fingerlike dorsal process. The trunk tip in both species functions as a refined eating tool. Directed examination of trunk tips of African elephants revealed the lack of postulated specialized mechanoreceptors, Meissner corpuscles,[19] confirming previous studies in the Asian species.

Detailed histological studies of the Asian trunk tip have demonstrated unique sensory innervation that resembles aspects of the sensory innervation of the mystacial skin of rodents or lip tissue of monkeys[38]. A plethora of sensory corpuscles and adaptations are present in the superficial dermis, including 1) an extremely high density of free nerve endings, often making intimate contact with the basal cells of the rete pegs, 2) numerous small convoluted multibranched corpuscles (MBC) that extend for long horizontal distances between several rete pegs, 3) many, very small, densely packed Pacinian (touch) corpuscles, and 4) two types of vibrissae, both innervated by hundreds of axons. Abundant regular vib-

rissae are present in the skin surrounding the trunk tip. Short vellus vibrissae that do not protrude from the skin surface are abundant in the fingertip.[38] Functionally this dense sensory innervation may be correlated with the tactile ability of elephants to use the trunk finger to grasp small objects for feeding or to insert chemically active and relevant samples into the ductal orifices of the vomeronasal organ for subsequent sensory processing.

## OLFACTION

### Main Olfactory System

Inhaled air travels up the long trunk, rapidly warmed so that the air reaching the turbinals is about 37°C (98.6°F). In this region a proportion of volatile molecules are diverted into a most unusual bony nasal cavity. Here the maxilloturbinals are almost totally absent; only the basal plate of the bony maxilloturbinal remains, forming the wall of the nasal tube.

The shape and directionality of the large (11 cm long), pear-shaped cribriform plate corresponding exactly to the outline of the olfactory bulb of the brain is in turn reflected in the more ventrally located ethmoid. "The part of the nasal cavity lying rostral to the ethmoid is of most peculiar appearance."[3] Unlike the three well-known nasal passages of most mammals, the elephant has only two passages, a narrow dorsal and more spacious ventral one.

The ethmoid is of imposing size, greater in breadth than in depth, occupying a strikingly large space. Uppermost is a shortened nasoturbinal forming a sharp boundary between the respiratory and olfactory region.[3]

In the African elephant the ethmoid possesses 7 medial endoturbinals, but because endoturbinal II is split there are 8 olfactory plates. All the ethmoturbinals, in only two rows, have much secondary folding[29]. For the Asian elephant, the ethmoid contains 7 endoturbinals and 32 ectoturbinals, with the latter on several rows.[43] In this species are many, varied anatomical extensions. Because the skull of the elephant changes directionality dramatically with maturation, the number of ectoturbinals increases and the configuration changes,[3] rendering the adult elephant an incredibly macrosmatic mammal.

In both species of extant elephants, especially the Asian species, the immensity and complexity of the more than several dozen turbinals is awesome. In contrast, other highly macrosmatic mammals, such as spiny anteaters, wallabys, Virginia opossums, bears, and foxes, have only, respectively, 12 endoturbinals, a wide, long nasoturbinal with two rows of 5 ethmoturbinals, three rows of 5 ethmoid turbinals, two rows of 7 turbinals, and two rows of 5 turbinals with extensions into the frontal and sphenoidal recesses.[30]

Interestingly, in the African elephant, the olfactory area interconnects with 9 sinuses.[29] Although olfactory receptors were not detected in the mucous membrane of one of these sinuses (Rasmussen, unpublished), further examination of these sinuses for olfactory tissue would be appropriate. Presumably, in elephants olfactory neurons with appropriate receptors function similarly toward odorants as described for other mammals by Buck—i.e., in a one odorant–one receptor scenario, with a particular neuron expressing only one type of odorant receptor[5]. Subsequently axons from odorant-specific neurons converge on similar glomeruli in the large main olfactory bulb.[33] Axonal projections and synaptic connections then relay and integrate olfactory information to the olfactory cortex in the ventral lateral brain, chiefly the pyriform cortex, the entorhinal cortex, and the olfactory tubercle.[1]

### Vomeronasal System

Elephants have a second, extensive olfactory system, the VNO complex. In most mammals the VNO is located above the hard palate with short ducts leading to the oral cavity. Similarly, in the 154-day fetal African elephant the VNO opens directly into the oral cavity with only a minute duct. However, by day 210 the African fetus has well-developed vomeronasal glands and the VNO ductal system has begun to acquire system features similar to the adult Asian elephant.[15,36] After birth and throughout the first several decades, craniofacial morphogenesis occurs during which cranial bones grow disproportionally to accommodate the tusks, concurrent with development of enormous cranial sinuses. As studied in the Asian species, the adult elephant possesses a very large paired tubular VNO, (each 1–2 cm in diameter and 12–15 cm in length) with millions of neural receptor cells in the concave medial portion.[21,36,43] Other unique features include paired ductal orifices in the dorsal palate and long paired ducts lined with stratified squamous epithelium. The ducts initially course anteriorly and then turn posterior and course laterally at a 45° angle, before returning to the midline where they merge prior to reaching the VNO. These ducts are between 15–20 cm long and are continuous with VNO.[36,43] Axons from the VNO receptor neurons presumably proceed to the accessory olfactory bulb (this structure has not been defined in elephants) and hence onward to connections in the amygdala, which in turn is connected with the medial preoptic and medial hypothalamic regions.[50]

### Transport-of-Pheromones Function of the Trunk Tip

Fascinatingly, the trunk and its tip play important facilitating functions prior to sensory reception of pheromones and other chemical signals in the VNO proper.[47] For example, a mature male Asian elephant in determining the preovulatory status of a female first sniffs a puddle of her freshly voided urine, and then, after dipping the trunk tip into the puddle (a check response), physically transports urine droplets, containing the phero-

mone, *Z*-7-12:Ac precisely to VNO ductal orifices in a response termed a *flehmen*. Concurrently, there is a biochemical transfer, effected by the acidic pH of the truncal mucus streaming out of the trunk onto the urine. Expelled female urine at the periovulatory phase contains *Z*-7-12:Ac bound to urinary albumin, but acidic conditions effect the squeezing-like release of *Z*-7-12:Ac, causing excess amounts to be sequestered by a truncal odorant binding protein, and the freed pheromone is placed on the duct orifices of VNO. The once-freed pheromone now gets caught by a third protein, facilitating the journey of the lipophilic substance through the somewhat aqueous and highly acidic mucus.

### Receptor Function of the VNO: Pheromones and Other Chemical Signals

Responses to the main olfactory and vomeronasal organ help elephants to discriminate individuals, relationships, and intrasexual sexual states and status. Urine especially provides a multitude of intra- and intersexual conspecific messages.[51,53] In addition, elephants apparently combine olfaction and VNO reception during responses to 1) interdigital areas for physiological state determination,[24] 2) temporal gland secretions for sexual state and status recognition and for metabolic and health assessments,[44,48] and 3) breath for musth state recognition.[41] Because behaviors and chemosensory responses are so clearly seen and can be quantitated, and because the tools of molecular biology are available, deciphering the dual roles of these two olfactory systems may soon be possible.

## MUSTH AND CHEMORECEPTION

### Anatomy of the Temporal Gland

The temporal gland, a cheek gland unique to mammoths and elephants, is a modified apocrine sweat gland releasing liquids of dual origin (glandular secretions and ultrafiltrates from blood).[11,12,44] The gland has well-described chemical communicative functions in Asian male elephants; usually only males in musth release secretions.[46,48] Asian females may secrete sporadically during pregnancy, labor, stress, and illness[35]. Although a chemocommunicative function was first postulated for the African species 40 years ago[6] and several chemical studies have identified unique farnesols and sesiquiterpenes,[16,17,56] only a few scientific studies of the secretions linked to behavioral effects have been published for this species.[6,18]

### Chemical Communication During Musth in Asian Elephants

In contrast, there are numerous chemical and behavioral studies of males of the Asian species. Initial work focused on the relationship between serum and TGS androgen concentrations in comparison to the chemical composition of TGS.[8,34] More recently, the relevance of the maturation status of the male Asian elephant to the chemical composition of the TGS secretion and its correlated observations made by ancient Hindus and modern elephant experts with precise chemical analyses of TGS from young maturing and older, fully mature male elephants demonstrate that young males secrete mellifluous exudates, smelling like honey, whereas older male secretions are malodorous.[46] Contained in the foul-smelling secretions of older males only is an acrid ketal, frontalin, demonstrated to act as a pheromone, eliciting differential responses from old males, young males, and females in various reproductive phases.[48] The physiology of the older males changes significantly during the musth episode,[44] in part explaining the higher proportion of bioactive ketones, such as cyclohexanone,[32] released in the TGS and in the breath.[41] Similar compounds are contained in the urine of musth males and may play a role in the preference of preovulatory females for older musth males with high serum testosterone.[51]

## GUSTATION (TASTE)

### Anatomical Aspects

The oral cavity of elephants contains taste detection structures both in the posterior, soft palate dorsal epithelium and on the tongue. The tongue itself is large, thick, and somewhat round, highly muscular and vascular, fitting in a groove formed by the lower jaws. The anterior end, about 7 cm long in adult African elephants, although free, is not as mobile as the posterior (base) end.[28,52] It is both an organ of touch and of taste.

The anterior two-thirds of the mucous membranes of the tongue are innervated by a collateral branch of the facial nerve, the chorda tympani.[27] The mandibular branch of the trigeminal nerve has a lingual nerve component that unites with the chorda tympani nerve and then detaches a mandibular branch and supplies other parts of the tongue.[27]

From posterior (base) to anterior (tip) of the tongue, the following gustatory structures are described. Anatomically the papillae of the tongues of African and Asian elephants appear similar, with 2–6 large circumvalate papillae on the dorsum of the tongue in front of the epiglottis and posterolaterally 18–30 vertical laminae or ridges, sometimes termed *Mayer's organ,* with intervening mucous-filled slits representing foliate papillae.[13,22,28] In the elephant these foliate papillae have a more anterior location than in other mammals. Kubota[22] described wartlike papillae located anterior of the foliate papillae, and laminated Pacinian-type corpuscles are spread over the whole tongue surface.

Kubota[22] states that the "vallate and foliate papillae have no taste buds" and that these papillae are only secretory in nature. However, for the circumvallate papillae electron microscopic studies have demonstrated an

unusual duality of taste buds with two taste buds per pair and an elevated basal membrane of each bud for the Asian species (Rasmussen, unpublished). The presence and location of taste pores on the elephant tongue has been demonstrated at the Riddle's Elephant Sanctuary by blue food dye applied to the tongue surface. Whether, unlike other mammals, the foliate papillae have no taste buds needs to be confirmed by histological investigation. Numerous taste buds have been described in the filiform papillae at the anterior tip of the tongue.[22] Fungiform papillae have not been described for elephants.

### Functional Aspects

In other mammals, receptors for various taste sensations are localized in regions of the tongue: bitter toward the rear, sour more laterally, sweet toward the tip, and salty overlapping the sweet tip region but also running laterally. All taste studies on elephants must be by inference, because electrophysiological recordings are not possible, or by behavioral responses. Many anecdotal observations reveal that elephants have a sweet tooth, easily detect certain sour and bitter substances, and are able to detect the protein content of crops and tannin and isothiocyanate content of barks. Our antifeedant studies with Asian elephants indicated that elephants are indifferent to certain usami and bitter substances, including quinine. The interesting question remains, as it does for humans, as to the extent that taste interacts with olfaction.

## DISEASES

Vascular changes in the tongue are seen in foot and mouth disease, and the blood vessels of the tongue lose their integrity during *Herpes* infections.

Disorders of olfaction, touch, or taste have not been reported except in cases of injuries. However, trunk injuries are reasonably common and a flaccid, irreversible trunk paralysis has been described in free-ranging African elephants.[23] There is a selective neuropathy of the truncal peripheral nerves, with myelin degeneration, endomyseal fibrosis, and subsequent muscle atrophy followed by compensatory hypertrophy. Trunk injuries in the wild are often a result of snares or explosives. Wild elephants have been seen with greatly shortened trunks, with damage to neuromuscular control of trunk, holes in the trunk or parts of the trunk fingers missing. For wild elephants, infections during such injuries may be lethal, but the severing of the entire trunk tip, up to 8 inches in length, may be nonlethal to captive elephants when followed by reconstructive surgery and/or intensive veterinary care and hand feeding by handlers[7] (personal communication, Roger Henneous, 1988). Interestingly, in both cases the elephants adapted to the shortened trunk, devising ingenious methods of feeding and in one case continuing to perform flehmen responses.

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# 33 Toxicology

Murray E. Fowler

## INTRODUCTION

Only a few instances of poisoning in elephants have been reported, and yet it is likely that they are susceptible to many of the noxious substances that affect domestic and wild animals. Numerous texts deal with the effects of poisonous substances in humans and domestic animals. The reader is directed to these sources for details of the diagnosis and treatment of specific toxicants.

Although examples of toxicities will be given and tables presented to list known toxic substances that may affect or have affected elephants, the primary focus will be discussions of basic concepts. Understanding concepts may make it possible to advise elephant managers on how to avoid poisoning by altering potentially dangerous situations.

Poisoning is often thought to be a wholly artificial phenomenon, which human beings have ultimate power to cause or prevent. This is a naive belief, because toxicants are as much a part of any free-ranging environment as are substances that nourish an animal. Adaptation to the ingestion of poisonous substances is a basic part of evolution. Just as a certain animal population may develop resistance to a microorganism, so do many animals develop tolerance to a specific toxicant.

Virtually nothing is known about the resistance of elephants to most toxicants. The death of free-ranging elephants is seldom investigated in any detail. Many poisonous substances leave little pathology upon which to make a diagnosis.

### Adaptation to Toxicants

Animals cope with toxicants through one or more of the following strategies: avoidance, dilution, degradation, or detoxification.[16] Should these strategies fail, the animal will be adversely affected and may ultimately die.

Avoidance is a crucial skill that must be learned early in life. It would be natural for elephants in their native habitat to avoid unpalatable (potentially toxic) plants unless driven by extreme hunger. An animal with a choice of plants may dilute a toxicant by ingesting only small quantities that fail to reach the threshold level for production of toxicity. A toxicant ingested by an elephant may be degraded within the digestive tract or excreted without being absorbed.

After the toxic agent is absorbed from the gastrointestinal tract, the body must either excrete it unchanged, sequester it in a nonactive storage site, detoxify it by molecular rearrangement, or suffer the ill effects caused by the toxicant. All vertebrates have developed general detoxification pathways that can deal with many different substances. Some have specialized mechanisms, unique to a given species.

Specific mechanisms are unknown in elephants. Much detoxification is carried out by hepatic microsomal enzyme activity. Enzyme systems require priming and periodic reactivation by exposure to nonlethal quantities of toxicants. Captive managed elephants may never be given an opportunity to stimulate these systems and may be at a great risk if suddenly exposed.

### Diagnosis of Poisoning

The clinical signs caused by most potential toxicants in elephants are unknown. Rarely will a diagnosis of poisoning be evident on initial examination. No pathognomonic signs of poisoning have been identified, and a limited number of pathognomonic lesions have been reported.

Diagnosis of poisoning depends upon analyzing a detailed history, evaluation of clinical signs, utilization of analytical procedures, and, in some cases, necropsy. The clinician must have some idea of possible toxicants in order to guide the laboratory in selecting appropriate screening tests. Possible disease conditions must be eliminated by a thorough examination and medical workup. It is imperative to keep detailed records of procedures and results.

It is unwise for a veterinarian to intimate casually, "It looks like a poison." A diagnosis of poisoning is not

valid if based solely on the premise that no other disease entity or cause could be determined.

### Treatment of Poisoning

The basic principles of treating a suspected case of poisoning include removal of the source and removal of the toxicant from the animal. If the toxicant has been applied externally, the animal must be bathed. This is difficult with elephants, but copious amounts of water should be used to attempt to rinse the toxicant from the surface of the skin.

A limited number of general and specific antidotes are available. General antidotes include 20% calcium gluconate, 10% glucose, and 10–20% sodium thiosulfate. Specific antidotes include calcium versenate against lead, sodium thiosulfate and sodium nitrite against cyanide, and atropine against anticholinesterase compounds used as insecticides or parasiticides. Activated charcoal is a general absorbent for some alkaloids and other toxicants.[18]

Early in the course of most poisonings, symptomatic and supportive therapy should be instituted. This will be of benefit in infectious or metabolic disease as well. Maintaining hydration with fluids, supporting respiration with oxygen and circulation with steroids and cardioactive drugs, and controlling central nervous system (CNS) stimulation with diazepam are indicated.

### Prevention of Poisoning

Three basic concepts are important in the prevention of poisoning in elephants.[15,17] The most important is elimination of exposure to toxicants. Purchased feeds should be carefully inspected for quality and the presence of weeds or foreign material. Processed feeds must be of the highest quality. Because it is impossible to inspect certain types of processed feeds for poor or toxic ingredients, the integrity and quality control of the supplier is of primary importance.

The second concept is avoidance of stress that enhances toxic effects. The third concept is provision of sufficient quantity and quality of nutrients in the diet to sustain healthy populations of gastrointestinal microflora and microfauna.

## CLASSES OF POISONS

Poisons that elephants may encounter include insecticides, rodenticides, disinfectants, cleansing agents, paints, antifreeze, plant toxins, mycotoxins, medications, and animal venoms. Table 33.1 provides a short list of potential poisons in elephants. Specific examples follow.

### Insecticides

Elephants are probably as susceptible to insecticides as other livestock species. A case of suspected organic phosphate insecticide poisoning has been reported. The clinical signs observed were typical of organic phosphate poisoning (depression, salivation, miosis, and diarrhea), but there was no muscle fasciculation. A study on serum cholinesterase has been reported.[40]

Demecron (organic phosphate insecticide) was confirmed as the toxic agent in the death of at least one elephant in Guwahati, India, and suspected in the deaths of nine other elephants in the same region.[7] The source of the insecticide was not established, but was thought to be the work of persons attempting to prevent crop raiding and building destruction. Elephants have an affinity for alcohol. It was felt that locals mixed the insecticide with country-made liquor and placed it where elephants could access it.

The conflict between elephants and villagers is likely to become more intensified with human population expansion and elephant habitat destruction, so officials may expect more poisoning cases to occur.

Administration of atropine sulfate is the standard therapy for organic phosphate and carbamate insecticide poisoning.[23] Poisoned animals are resistant to the effects of atropine, so the initial dose should be 0.2 to 0.4 mg/kg rather than the recommended elephant dose of 0.01 mg/kg IV. If possible, half the dose should be given intravenously and the rest subcutaneously. Activated charcoal should be administered orally at 1 to 3 g/kg if the organic phosphate was taken orally.

### Rodenticides

Only one case of rodenticide poisoning in elephants has been reported in the scientific literature, but these agents affect a broad host range, and elephants are likely to be susceptible if exposed. Owners should be cautioned about the use of rat and mouse bait in impregnated grain that may be placed where elephants may gain access.

Sodium fluoroacetate (1080) was used maliciously in a zoo in Sao Paulo, Brazil (personal communication, Dr. Sandra Corrêa, May 2005). Three chimpanzees, one orangutan, three tapirs, three camels, and an elephant were found dead over a 2-week period. Sodium fluoroacetate disrupts the Krebs cycle and causes convulsions, vomition, involuntary urination and defecation, respiratory paralysis, muscle tetany, and rapid death without producing any gross lesions.

Zinc phosphide poisoning was diagnosed in zoo elephants in Malaysia, probably from intentional poisoning. Zinc phosphide is hydrolyzed to highly toxic phosgene gas in the acid milieu of the stomach. If a diagnosis could be made early enough, a stomach lavage with 5% sodium bicarbonate would aid by increasing the pH of the stomach contents.[6]

### Heavy Metals

Lead, arsenic, mercury, copper, and molybdenum are used in agricultural products and paints. It should be assumed that elephants are susceptible. Clinical signs and lesions are presumed to be similar to those of poisoned

**Table 33.1.** Reported and Potential Nonplant Toxicity in Elephants

| Toxicant | Clinical Signs in Animals | Sources | Diagnosis | Pathology | Management |
|---|---|---|---|---|---|
| Arsenic (As) | Colic, weakness, trembling, diarrhea, dehydration, depression | Contaminated forage, pesticides, herbicides | Analysis of liver, kidney, stomach contents | Gastroenteritis | Routine toxicologic case management regimen |
| Copper (Cu) | Anorexia, icterus, hemoglobinuria, increased respiratory disease prevalence | Insecticides, feed supplements | Analysis of blood and liver Cu levels for elephants | Hepatic necrosis, anemia, evidence of hemolysis | Prognosis is grave; blood transfusions, antibiotics |
| Lead (Pb) | Depression, colic, diarrhea, ataxia, circling, blindness | Contaminated forage, lead-based paint | Analysis of blood, liver, kidneys | Encephalopathy, gastroenteritis | CaEDTA (110 mg/kg IV for 5 days) |
| Sodium chloride (NaCl) | Cerebral edema causes convulsions, blindness, partial paralysis | Ingestion of a pelleted feed containing 9.29% NaCl due to a manufacturing error, (Stehlik 1971, 1974) | Signs, lesions, and analysis of feed Plasma Na >150 mEq/l; CSF Na >145 mEq/l; brain Na >1800 ppm | Cerebral edema | Poisoning usually a combination of high sodium intake and water deprivation or dehydration |
| Sodium fluoroacetate (1080) | Sudden death, cardiac arrhythmias, trembling, colic convulsions | Rodenticide, intentional addition of agent to feed | History, signs, analysis of stomach contents, liver, and kidney | No lesions | Nothing specific; prognosis is grave, with death in minutes from time of ingestion of a toxic dose |
| Organophosphate/ carbamate insecticides | Salivation, colic, diarrhea, dyspnea, miosis, twitching, tetany, depression | Improper dose of parasiticide, drinking from an abandoned livestock dipping tank | Analysis of blood cholinesterase activity | No lesions | High dose of atropine,(0.4 mg/kg +) |
| Organochlorine insecticides | Hypersensitivity, muscle fasciculation, depression, tonic/clonic convulsions | Improper use of insecticides | History, signs, analysis of stomach contents, liver, and kidney | No lesions | Routine toxicologic case management regimen |
| Strychnine | Tenesmus, tetany following stimulation, and then depression | Access to rodent bait, intentional poisoning | Analysis of stomach contents | No lesions | Sedation and anesthesia, respiratory support |
| Anticoagulants | Hemorrhages, external and internal | Access to rodent bait | Clinical pathology findings, prolonged clotting time | Hemorrhages | Blood transfusion is the only specific treatment, although vitamin K is also administered |

horses, cattle, sheep, and goats. A study in Africa established lead levels in elephants.[11]

**Copper poisoning.** Absorbed copper is stored in hepatocyte lysosomes. When the storage capacity of the liver is exceeded, copper is released into the cytoplasm of the hepatocyte, causing hepatocellular necrosis. After the hepatocyte cell wall has been destroyed, copper is released into the circulation causing hemolysis, icterus, and anemia. The excessive amount of free hemoglobin in the circulation may obstruct renal tubules, resulting in renal failure.[18]

Elephant managers should be advised that pellets or mineral mixes intended for cattle are not suitable as supplements for elephants unless the total dietary intake of copper, including the supplement, does not exceed 15 mg/kg of feed. It is also necessary to maintain a copper/molybdenum ratio <10:1.

Clinical signs. Signs of copper toxicity are similar in all species of animals. The animal becomes lethargic and stops eating. This may be followed by recumbency, marked depression, anemia, pale mucous membranes, dyspnea, and hypothermia. Hemaglobinuria and icterus may or may not be present.

Diagnosis. At necropsy, hepatomegaly is the primary gross lesion and hepatocellular necrosis, bile duct prolif-

eration, and hemoglobin impaction of renal tubules the histologic lesions. Serum enzymes associated with hepatic necrosis are usually elevated. Serum copper levels remain within normal limits until hemolysis and hepatic necrosis occur.

An animal may die from either hepatic insufficiency or renal failure.

Management. No treatment is effective after hepatic necrosis and hemolysis occur. Blood transfusion has been recommended, but in elephants this is not likely to be possible, and it won't counteract renal tubular impaction.

### Carbon Monoxide Poisoning

It may be difficult to visualize how elephants could be exposed to carbon monoxide (CO). It is unlikely that peracute or acute toxicity would develop, but Ball makes a case for possible chronic toxicity as a cause for the high prevalence of fetal death and perinatal mortality in captive elephants that are kept in heated elephant houses for several winter months.[2]

Carbon monoxide toxicity caused by inhalation of exhaust fumes while being transported has caused problems in moving livestock, but a study of transporting elephants found no evidence of CO buildup.[43]

**Clinical signs.** Carbon monoxide molecules replace the oxygen molecule normally transported on hemoglobin (Hb). The bond COHb is firm, and when CO ties up 40% of the Hb, stupor, coma, and death may ensue.

Experimental evidence in domestic animals has shown that a level of 9% COHb in maternal circulation will effectively reduce fetal oxygen blood content by 21%, equivalent to a 41% loss of hemoglobin or blood flow.[26] Fetal tissue is particularly sensitive to hypoxia, and sublethal levels of carbon monoxide may produce teratogenicity, neurologic disorders, reduced birth weights, and an increased prevalence of stillbirths.[27]

**Management.** Elephant managers should ensure that heating units in elephant houses are functioning properly and that there is adequate ventilation in the facility.

### Alcohol

Elephants are attracted to ethyl alcohol. Free-ranging African elephants eagerly consume the ripened fruit of the Marula tree *Sclerocarya birrea* when it falls to the ground. The fruits quickly begin to ferment and when eaten by elephants, "drives them mad." Affected elephants are described as being drunk. Marula trees have been used extensively for thousands of years in Africa for food and herbal medicines. Elephants in Asia are reported to favor durian fruit *Durio zebethinus*, perhaps for the same reason (personal communication, Dr. Susan Mikota, May 2004).

Villagers in Southeast Asia have mixed powerful insecticides in homemade liquors or rice beer and placed containers where elephants could drink. News reports indicate that elephants have killed villagers during drunken rampages.

A clinical trial was conducted on the effects of alcohol in elephants. Signs appeared in 30 minutes and included decreased feeding and drinking, increased ear flapping, and increased swaying and rocking with the eyes closed.[36]

A zoo elephant that became ataxic in Denmark was thought to be "drunk." However, a later diagnosis of ryegrass staggers was made.[22]

### Miscellaneous Toxins

Other toxins reported in elephants include paint solvent[31] and salt (NaCl).[38,39]

## PLANTS

Poisonous plants present special problems for elephants. Free-ranging young elephants quickly learn from their mother or allomothers (aunts) the proper plants to eat. If they consume a small quantity of plants that contain various secondary plant compounds, the elephant's metabolic processes are stimulated to develop detoxification pathways. They are in essence, "priming the pump" so that if they consume more of the plant in the future their system can deal with it.[16]

Young elephants that have been captured in India and Southeast Asia have likely developed those habits that help them to avoid toxic plants. Elephant calves that have been captured from the wild and placed in zoos or calves born to cows in captivity may never be taught how to avoid toxic plants.

Elephants are fastidious eaters and usually avoid plants with a bitter taste, but hungry elephants may eat them. This may be the situation that exists in elephants in Asia that are in an urban environment and severely restricted on feed intake.

Plant poisoning is often an environmental mistake. Animals newly introduced into an environment may investigate any plants growing in or near the enclosure. Elephant managers must also be aware of trees and shrubs that may be within reach of an elephant's trunk. One author feels that soil consumption by elephants may help minimize the effects of secondary plant compounds found in forest browse.[24]

Changes in the environment may cause an elephant to consume a strange plant, or even a plant that has been present all along, but was previously avoided. A tragic example of such a situation occurred at a Safari Park where the author was consulting. It was noted that numerous tree tobacco *Nicotiana glauca* plants were growing in a large enclosure containing giraffes. The author advised the manager to remove the plants to avoid the risk of poisoning. The reply was, "We don't need to worry about that plant because it has always been there

and the giraffes don't touch it." Two months later an article appeared in the newspaper that four giraffes had died at that park from tree tobacco poisoning.

Managers of elephants in zoos, Safari Parks, and circuses constantly need to be aware of plants in the elephants' environment and have strict control over what is being used for browse. As an example of what might happen, the author once observed some workmen cutting browse to feed animals in a large zoo. To the author's horror, a workman started collecting branches of yellow oleander *Thevetia peruviana*. When questioned about his actions, he said, "I thought it was podocarpus *Podocarpus* spp."

Only a few reports of plant poisoning in elephants are known. There are hundreds of plants that would be undesirable for elephants to consume. None has been evaluated in terms of the susceptibility of elephants to the toxins and the amounts of the plant required to produce toxicity. There is reason to believe that plants having a toxin with broad species susceptibility are dangerous if elephants consume them.

It is neither prudent nor possible to list every potential poisonous plant in every country or region. A selected list is found in Table 33.2. A list of common names for poisonous plants is found in Table 33.3. A wide variety of plants may be found in many locations in the world, either as native species or as ornamentals. Some have special climatic requirements, so they may not be ubiquitous even when found in a given region or country.

The author has also included a list of browse plants suitable for elephants in North America (Table 33.4). The author is not qualified to make such a list for other regions of the world.

Numerous reference books on poisonous plants exist. Regional and country books may be found in Agricultural College libraries. The following are references: North America,[4] Australia,[12] Europe,[20] New Zealand,[32] India,[9] United Kingdom,[10] and Africa.[25] None of these books discuss plants that are toxic to elephants. The best alternative is to consider plants known to poison horses because elephants and horses have similar gastrointestinal anatomy—namely, a simple stomach and hindgut fermentation.

Ornamental plants may be a special hazard for circus and zoo elephants, especially if elephants can reach them. People have an innate desire to feed animals in a zoo and may pick leaves from ornamental plants to feed to the elephants. Ride elephants may inadvertently be placed in a location with access to ornamental plants. Unfortunately, ornamental plants may not be listed in poisonous plant lists and booklets of the region, because many ornamentals are not native. Handlers must learn to identify hazardous plants in the environment of their elephants.

A few plants are discussed in some detail in the following sections; others are listed in the tables.

## Rhododendron Poisoning

**The plants.** All members of *Rhododendron* spp., family Ericaceae, should be considered dangerous for elephants to ingest. These shrubs grow wild in northern temperate climates. They are also planted in zoos and public and private gardens around the world. Additional genera that should be considered suspect include Labrador tea (*Ledum* spp.), black laurel (*Leucothoe* spp.), and (*Kalmia* spp.), all of which are small shrubs.

Many genera and species within the plant family Ericaceae contain a resin, andromedotoxin, and a glycoside, arbutin, which produce an identical syndrome in a broad host range.[15,17,18]

**Signs.** Rhododendron leaves were mistaken for proper browse and fed to a group of 10 circus elephants.[34] An hour later all the elephants began to exhibit colic-like signs accompanied by intense restlessness and diarrhea. The signs diminished after 3 hours in most of the elephants, but signs worsened in one elephant that had blood in the feces. Ten hours later the signs recurred in another elephant, with colic becoming intense. That elephant collapsed and became recumbent and died within 24 hours. The clinical syndrome is similar to that produced by ingestion of oleander, which should be included in a differential diagnosis.

**Management.** Treatment of rhododendron poisoning is nonspecific. Activated charcoal is a general antidote and may be administered by stomach tube or as a drench consisting of a slurry of activated charcoal in water. However, such therapy is dangerous in a vomiting animal. Atropine (0.01 mg/kg) may be used to alleviate bradycardia. Other symptomatic treatment may be indicated.

## Cardioactive Glycoside Poisoning

**The plants.** Oleander, *Nerium oleander*, is one of the most toxic shrubs to which an elephant or any other mammal may be exposed. The lethal oral dose of either green or dried leaves is 25 to 50 mg/kg. Poisoning is usually caused by ingestion of dried leaves because the green leaves have a distinctive bitter taste. The poisonous principles are cardioactive glycosides, similar in action to digitalis (oleandrin and neriantin).

Oleander is a beautiful ornamental shrub, grown extensively throughout California and along the southern tier of the United States. Unfortunately, it has also been planted in and around zoos, or in areas where circus animals may be walked or maintained. As a potted shrub that can be moved inside during the winter, it may be found almost anywhere in the world.

Another plant, *Cryptostegia grandiflora* (family Asclepiadaceae) caused the death of two young African elephants when they slipped under an electric fence surrounding a home on a game preserve and ate the ornamental plant.[3]

**Table 33.2.** Selected Poisonous Plants That May Have or Have Affected Elephants

| Common Name | Scientific Name | Poisonous Principle | Distribution/ Habitat | Signs of Poisoning | Plant Characteristics |
|---|---|---|---|---|---|
| Oleander | *Nerium oleander*<br>Family Apocynacae | Cardioactive glycoside | Native to Mediterranean, ornamental in many subtropical and temperate regions | Diarrhea, colic, cardiac irregularities, cyanosis | Large linear leaves with characteristic main central vein with numerous parallel secondary veins at a slight angle to the main vein |
| Yellow oleander, lucky nut, be-still tree, trumpet flower, Codo de fraile | *Thevetia peruviana*<br>Family Apocynacae | Cardioactive glycoside | Common ornamental in subtropical and temperate climates | Diarrhea, colic, cardiac irregularities, cyanosis | |
| Rhododendrons/ azaleas | *Rhododendron* spp.<br>Family Ericaceae; Toxic genera Ledum, Leucothoe, Kalmia Pieres | Andromedotoxin (diterpene arbutin) | Northern hemispheres, in north cool, temperate climates, present in some locations in India | Vomiting, colic, paresis, anorexia, muscle twitches | Shrubs with showy flowers; commonly planted as an ornamental |
| Tutu | *Corikaria arborea*<br>Family Coriariaceae | Tutin, Picrotoxinlike alkaloid | New Zealand; other species grow in Eastern Asia, Central, and South America, Mediterranean | Excitement, ataxia, salivation profuse, watery diarrhea, trembling, convulsions, prostration | Shrub to small tree, leaves simple and opposite |
| Black locust, false acacia | *Robinia pseudoacacia*<br>Family Fabaceae | Alkaloidal | Commonly grown as an ornamental tree throughout the world | Diarrhea, collapse, shock | Small to large tree; a legume with moderate-sized brown pods at maturity; white pealike flowers |
| Yew, Japanese yew, English yew | *Taxus* spp.<br>*Taxus baccate*<br>Family Taxaceae | Alkaloidal | Common garden ornamental shrub or small tree throughout the world | Sudden death, dyspnea, diarrhea, collapse | Conifer; fruit is a red pulp surrounding a single seed |
| Wild tobacco, tree tobacco, commercial tobacco | *Nicotiana* spp.<br>Family Solonaceae | Nicotine alkaloids, anabasine | Tree tobacco grows wild in waste places, common garden ornamental | Stimulation of CNS, and then depression, sweating, muscle twitches, convulsions | Variable foliage; always has a showy tubular flower |
| European bittersweet, nightshades | *Solanum dulcamara, Solanum* spp.<br>Family Solonaceae | Steroid glycosides, solanine | Ornamental (bittersweet), weeds of waste places and in forage crops | Vomiting, diarrhea, colic, depression, weakness, cardiac arrhythmias, dyspnea, hemaglobinuria | Foliage variable; flowers large or small, but all with the corolla united and retroflexed; the 5 yellow stamens tightly surround the pistil |
| Thorn apple, jimsonweed, tolgaucha, devil's trumpet | *Datura stramonium, D.* spp.<br>Family Solonaceae | Atropine alkaloids, hyoscyamine, scopolamine | Found as weeds and small shrubs throughout the world; the seeds have been used to produce hallucinations in people | Bizarre behavior, weakness, tachycardia, mydriasis, dry mucous membranes, photophobia, mania, delirium | Foliage variable; flower is large with a tubular corolla; fruit is a thorny capsule containing numerous seeds; in a suspected poisoning in an elephant, it was treated with atropine, which is the toxic agent |
| Pokeweed, poke, pokeberry, inkberry, scoke, red ink plant | *Phytollaca americana*<br>Family Phytolaccaceae | Saponins, glycosides | | Mild to severe diarrhea, possible incoordination, convulsions | |

**Table 33.2.** Selected Poisonous Plants That May Have or Have Affected Elephants (*continued*)

| Common Name | Scientific Name | Poisonous Principle | Distribution/ Habitat | Signs of Poisoning | Plant Characteristics |
|---|---|---|---|---|---|
| Castorbean, palma Christi, castor-oil plant, higuerilla, wonder tree | *Ricinus communis* Family Euphorbiaceae | Ricin, a glycoprotein found primarily in the seed; has been used for bioterrism | Grown for its valuable oil and may escape and become a weed of waste places in many locations throughout the world | Severe watery diarrhea, fever early, elevated PCV, pounding heartbeat, death within 36 hours of ingestion | Has a characteristic large palmately lobed leaf; variegated seeds are in a thorny capsule |
| Horse chestnut, buckeye | *Aesculus hippocastanum, A.* spp. | Saponin/glycoside | | Sawhorse stance, incoordination, hypermetria, hyperesthesia, colic, mydriasis, trembling, depression | Leaves palmately compounded with 5 to 7 leaflets; shrubs to large trees |
| Cherry, peach, plum, wild cherry, chokecherry | *Prunus serotina, P.* spp. Family Rosaceae | Cyanogenic glycosides | Grown for fruit production, throughout the world; wild species vary from region to region | Sudden death preceded by severe dyspnea and convulsions | Shrubs to large trees; variable foliage; crushing the leaves usually produces an odor of bitter almonds |
| Chinaberry, pride-of-India, umbrella tree, Persian lilac, white cedar | *Melia azedarach* Family Meliaceae | A complex triterpene, epoxide meliatoxin; this plant is used in herbal medicine as a parasiticide | Native to Asia, but grown as an ornamental throughout the world | Increased salivation, retching, anorexia, diarrhea; excitement, ataxia, convulsions, paralysis | Large tree; pinnately compound leaves; fruits are yellowish drupes containing 1–6 seeds |
| Milkweeds, woolly pod milkweed, Mexican whorled milkweed | *Asclepias eriocarpa, A. fascicularis, S. syriaca, S. speciosa, S.* spp. | Steroidal glycosides; some species have cardiac activity, and others contain more neurotoxic agents | Primarily found in the New World, but some are found in Africa, and used for poison arrow potions | Colic, incoordination, posterior weakness, trembling, falling, mydriasis, profuse sweating, atony of intestines early, and then profuse diarrhea late | May be an annual forb, vine, or small shrub; flower is unique, designed to attract insects; fruit pod contains numerous seeds attached to silky filaments |
| Mycotoxicosis | Numerous toxins produced by fungal growth on forages; *Lolium perenne* | Alkaloidal mycotoxins, Lolitrem A, C.D. produced by the fungus *Neotyphodium lolii* | Most commonly causes disease in pasture, but may also be seen in hay | Severe ataxia in an elephant in Denmark (Groendahl-Nielsen 2004); a bull became ataxic 4 days after feeding moldy hay stopped | Ryegrass is a common pasture grass in the United States and elsewhere in the world; it is also harvested for hay |
| Blue-green algal bloom | *Microcystis* spp. | Various toxic agents | Buildup in stagnant fresh water | Sudden death, photosensitization | |

**Clinical signs.** The clinical signs of cardioactive glycoside poisoning are similar in all species of mammals, with sudden death being a prominent sign. Elephants may become anorectic and depressed and may lie down unless forced to rise. A major observable sign is frequent, projectile, catarrhal to hemorrhagic diarrhea. Signs of colic may accompany the diarrhea. If the ingested dose is high, the animal may die of cardiac complications before diarrhea develops.

The cardioactive glycoside has a direct effect on the cardiac musculature. Various conduction abnormalities may be heard on auscultation over a period of a few minutes because of the rate and rhythm change. There may be bradycardia, tachycardia, drop beats, and partial and complete blocks. Impaired circulation may result in cyanosis, muscle tremors, patchy perspiration, and dyspnea. Terminally, tachycardia progresses to ventricular fibrillation and agonal struggling. The signs in the two young elephants included weakness, dyspnea, colic, recumbency convulsions, and muscle tremors.[3]

**Diagnosis.** Auscultation of adult heart activity is not easily accomplished in an adult elephant. If the sick elephant is trained in either free contact or protected contact, have the handler move the left forelimb forward, or better still, have the elephant lift the left forelimb. A

**Table 33.3.** Common Names of Selected Poisonous Plants of Concern to Elephant Managers

| Scientific Name | Plant Family | Common Name North America | Common Name Africa | Common Name India | Common Name U.K./Europe |
|---|---|---|---|---|---|
| *Nerium oleander** | Apocynaceae | Oleander | Oleander, selonsroos (ornamental) | Laurier-rose, oleander, Difli | Oleander |
| *Thevetia peruviana* | Apocynaceae | Yellow oleander, lucky nut, be-still tree, trumpet flower, Codo de fraile | | Not reported | Not reported |
| *Taxus* spp.* | Taxaceae | Yew | Not reported | Not reported | Ibe, EJiben, Kantel-Baum, taxus |
| *Robinia pseudoacacia* | Fabaceae | Black locust, false acacia | | Not reported | Falsche Akazie |
| *Nicotiana* spp.* | Solonaceae | Tree tobacco, wild tobacco | Wild tobacco, wildetabak | Not reported | Bauerentabak |
| *Datura* spp.* | Solonaceae | Jimsonweed, Jamestown weed, thorn apple, tolgoucha | Thorn apple, stinkblaar | | Stechapfel, stechelnuss, dornapfel |
| *Solanum* spp.* | Solonaceae | Nightshade, deadly nightshade, European bittersweet | Nightshade, nastergal | | Bittersuesser Nachstachatten, Schwarzer Nachtaschatten |
| *Ricinus communis* | Euphorbiaceae | Castorbean, palma Christi, castor-oil plant, higuerilla, wonder tree | Castor-oil plant, kasterolieboom | Not reported | Not reported |
| *Melia azedarach* | Meliaceae | Chinaberry, umbrella tree, Persian lilac, white cedar | Syringa berrytree, seringboom | Neem, Margosa Tree, India lilac, Ceylon mahogany, pride-of-India | Not reported |

*Many species, which vary from country/locality.

pulse may be palpated on an artery on the ear pinna, but it would be difficult to evaluate conduction abnormalities. A more definitive diagnosis requires an electrocardiogram showing skipped beats and heightened T waves.

At necropsy, lesions are limited to enteritis plus petechial or ecchymotic hemorrhages of the gastrointestinal serosa or of the epicardium, endocardium, or pericardium. Stomach contents should be carefully examined for the presence of leaf segments. Oleander leaves have a unique parallel vein pattern off the prominent single midrib. Finding even a small segment of a leaf in the stomach contents would justify a diagnosis of oleander poisoning.

Diagnosis may be facilitated by 2D thin layer chromatography of gastrointestinal contents extracted with dichloromethane.[19] The oleandrin may have moved along the intestinal tract, so it is wise to sample the contents of the colon as well as the stomach. The technique has been applied to urine, but is not as sensitive as with digestive tract contents. Equine feces has been used for antemortem diagnosis in more chronic cases.[19]

## Yew Poisoning

**The plant.** Yew *Taxus* spp. have been known to be highly toxic to farm animals and humans for centuries. Yews are common ornamentals, but should not be planted at a site where elephants may reach them. Various species of yew may contain as many as 10 different alkaloids; some are nontoxic and others are extremely potent poisons. Taxine has also been shown to have anticarcinogenic activity.

**Clinical signs.** Death may be so rapid that signs may not be observed. The potent toxins affect the central nervous system, causing vomition, tremors, convulsions, and dyspnea in humans and farm animals.

**Diagnosis.** No lesions are produced. Unless a history of ingestion is forthcoming, the only diagnostic aid would be to find the characteristic needles in the stomach contents.

**Management.** There is no specific treatment for yew poisoning, but supportive therapy is indicated.

## Kodo Millett Poisoning

**The plant.** Kodo millett (Vargu) *Paspalum scrobiculatum* has been reported to be responsible for the deaths of 14 free-ranging elephants in India.[29] The elephants were found dead or moribund in rice fields or in an adjacent forest preserve.

**Table 33.4.** Trees, Shrubs, and Grasses That May Be Used for Browse for Elephants in North America*,**

| Common Name North America | Scientific Name |
|---|---|
| Maples (except red maple), boxelder | *Acer* spp. |
| Ailanthus, tree of heaven | *Ailanthus altissima* |
| Silk tree, mimosa | *Albizia* spp. |
| Alders | *Alnus* spp. |
| Birches | *Betula* spp. |
| Hornbeams | *Carpinus* spp. |
| Hickory, pecan | *Carya* spp. |
| Catalpa, Indian bean | *Catalpa* spp. |
| Hackberry | *Celtis* spp. |
| Redbud, Judas tree | *Cercis* spp. |
| Dogwood | *Cornus* spp. |
| Hawthorn | *Crataegus* spp. |
| Persimmon | *Diospyros* spp. |
| Russian olive | *Elaeagnus augustifolia* |
| Beeches | *Fagus* spp. |
| Fig trees | *Ficus* spp. |
| Ashes | *Fraxinus* spp. |
| Honey locust | *Gleditsia* spp. |
| Acacias (some may be poisonous, know the species) | *Acacia* spp. |
| Jacaranda, green ebony | *Jacaranda* spp. |
| Crape myrtle | *Lagerstroemia indica* |
| Larch | *Larix decidua* |
| Sweetgum, liquidambar | *Liquidambar styraciflua* |
| Tulip tree, yellow poplar | *Lirodendron tulipifera* |
| Magnolia, cucumber tree | *Magnolia* spp. |
| Mulberry | *Morus* spp. |
| Crabapple | *Malus* spp. |
| Pistache, Chinese pistache | *Pistachio chinensis* |
| Plane tree, sycamore | *Plantanus* spp |
| Poplar, cottonwood, aspen | *Populus* spp. |
| Mesquite | *Prosopis* spp. |
| Pear, flowering pear | *Pyrus calleryana* |
| Willows | *Salix* spp. |
| Linden, basswood | *Tilia* spp. |
| Elms | *Ulmus* spp |
| Eugenia | *Euginia* spp. |
| Bottlebrush | *Collistemon* spp. |
| Bamboo | *Bambusa* spp. |
| Heavenly bamboo | *Nandia domestica* |
| Giant reedgrass | *Calamovilfa gigantea* |
| Most hay and pasture grasses are fine, but use caution in feeding wilted or frosted Johnson grass *Sorghum halpense* and sudan grass, *Sorghum vulgare sudanensis* | |

*= Palatability for elephants may vary considerably.
**= This is not an exhaustive listing.

Paspalum is a large genus of 300–350 species of grasses primarily found in the New World. Well-known forage grasses in the United States include dallisgrass *Paspalum dilatatum* and Bahiagrass *P. notatum*. In certain years dallisgrass seed may become infected with a fungus *Claviceps paspali* that produces a mycotoxin similar in action to ergot.

**Signs.** Signs in livestock include nervousness; tremors of the lips, face, and ears; head nodding; incoordination; and convulsions. Most livestock recover unless they fall into awkward places or into water and drown. No signs have been described in elephants other than prostration and death.

**Diagnosis.** Diagnosis is made by access to mature grain of kodo millett, which contains sclerotia of the fungus or analysis for the mycotoxin.

**Management.** Management includes protecting the elephant from injury and providing feed and water if the animal is recumbent.

## Datura Poisoning

**The plant.** Thornapple *Datura stramonium* is found throughout the world. The toxic agents include atropine and atropinelike alkaloids (scopolamine, hyoscine).

**Signs.** Two Asian elephants were taken to an area of wasteland in a New Zealand zoo to graze and exercise under supervision.[13] An hour later, one elephant was reported to be blind and was stumbling and falling. A few minutes later it was depressed and the trunk was limp.

**Diagnosis.** The wasteland was inspected for poisonous plants and it was noted that two plants of *Datura stramonium* were missing and presumed eaten. As a complicating factor, poison hemlock *Conium maculatum* plants were also missing and presumed eaten. The clinical signs and the rapidity of exhibiting signs are indicative of datura poisoning and not conium.

**Management.** Caregivers, not realizing that the toxic agent in the plant was atropine, administered 16 milligrams of atropine (0.005 mg/kg). That low dose probably did not exacerbate the problem, but if atropine had been administered in a therapeutic dose the results could have been disastrous.

Sedation to control central nervous system stimulation would be the appropriate therapy.

## Tutu Poisoning

**The plant.** Tutu *Coriaria arborea* (family Coriariaceae) caused nonlethal poisoning in two circus elephants in New Zealand. The elephants were being transported in an open-sided truck, giving them access to the leaves of the tree.[1]

**Signs.** Signs began 3–4 hours following ingestion and included trembling, profuse salivation, and diarrhea. Early signs led to generalized shaking, loss of balance, falling, paddling, coma, dyspnea, and periodic convulsions.

**Diagnosis.** The elephants were observed eating the foliage. Signs were not pathognomonic. A differential diagnosis would have to include insecticide poisoning and cyanide.

**Management.** One elephant that weighed 4000 kg was sedated with 2.25 kg of magnesium sulfate orally, 2.5 g promethazine hydochloride, and 2.5 g chlorpromazine IM. Later, 8 g of pentobarbitone sodium was administered IV and repeated as necessary. The second elephant did not receive magnesium sulfate, but was similarly sedated. Both elephants were recumbent for 17 h and 26 h, respectively, but they recovered.

## ARROW POISON

Native societies in many countries have a long history of using poisoned arrows to collect animals for food (including elephants), raiment, and intertribal warfare. The substance used to poison the animal was a closely guarded secret and most of the concoctions were a complex mix of plant and animal products.[42]

Poisoned arrows and some of the products used were the forerunners of chemical immobilization drugs that have become essential for managing the health and well being of wild animals, both in captivity and in the free-ranging state. South American native societies used plant material with curare activity. Various curare derivatives were used in the early days of chemical immobilization. A compound of nicotine alkaloids was the first drug sold as an immobilizing agent in the United States in the 1950s.

In native countries the selection of plants to be used was dictated by custom and the species available locally. See Table 33.5 for a listing of plants that were commonly used. Some tribes derived an income from selling their highly effective potion to other tribes. Plant and animal toxins had to be heat and desiccation stable in order to remain toxic during boiling or heating over a fire followed by prolonged drying.

The precise preparation method may not be known to outsiders, but in general was followed as described for *Strophanthus* spp. in Africa. Seeds were collected, crushed, and mixed with saliva to form a paste, which was left to dry in sunlight for several hours.

All parts of *Acokanthera* spp. are poisonous, so broken-up leaves and stems plus wood chips were placed in a large pot of boiling water and heated for up to 12 hours. When all the water was evaporated, a sticky black residue was left. The plant parts were discarded and the residue cut into pieces that may be wrapped and stored for later use or to sell. The residue was rehydrated to form a paste that was applied to the arrow shaft just behind the arrowhead. Frequently, the milky latex from *Euphorbia* spp. was added to the concoction to help it stick to the arrow shaft.

Animals contributed to potions used for poison arrows. The Kalahari bushmen (Sans) collected the larvae (grub) and pupae of chrysomid beetles (*Diamphidia* spp. and *Lebistina* spp.).

The skin of poison arrow frogs of South America (*Dendrobates* spp. and *Phyllobates* spp.) contains glands that secrete highly toxic cardioactive glycosides, whose effect is ultimately to cause ventricular fibrillation. Native societies in restricted areas of Columbia in South America captured the tiny colorful frogs. Fresh frog skin was gently heated over a flame to stimulate the drip of a secretion which was collected, dried, and then applied to the tip of darts. The darts were propelled by using a

**Table 33.5.** Plants Used for Arrow Poisons

| Scientific Name | Common Name | Plant Family | Toxic Constituent | Part of the Plant Used | Where Used |
|---|---|---|---|---|---|
| *Strophanthus kombe, S.* spp. | Poison rope, giftou | Apocynaceae | Cardioactive glycosides, Strophanthin | Seeds | Tropical Africa, some species grown in Asia and SE Asia |
| *Acokanthera* spp. | Bushman's poison bush, giftoon, boesmangif, poison arrow plant, wintersweet | Apocynaceae | Cardioactive glycosides, Ouabin, acaokantherin | All parts, including wood | Africa |
| *Euphorbia ingens, E.* spp. | Candelabra tree, gewone naboom | Euphorbiaceae | Sticky, milky latex | Latex exuding from plant when cut | Africa |
| *Adenium boehmianum* | Desert rose, mock azalea, kudu lily, desert azalea | Apocynaceae | Cardioactive glycoside | Leaves and stems | Africa, primarily to help the potion stick to the arrow |
| *Ascelpias stellifera* | Milkweed, melkbos | Asclepidaceae | Cardioactive glycoside | Leaves | Africa, not a major constituent |
| *Strychnos toxifera* | Curare plant | Loganiaceae | Alkaloids, calabash curare, curarine | Young bark scrapings | South America |
| *Chondrodendron tomentosum* | Curare plant | Menispermaceae | Alkaloids, tube curare, tubocurarin | Young bark scrapings | South America |
| *Spondianthus preussii* | Obubili | Euphorbiaceae | Monofluoracetic acid, oxalic acid | Leaves | Used by poachers in Central and West Africa |

tube made of bamboo strips (blow pipe). This concoction was obviously not used on elephants.

The potency of plant and animal concoctions could be extremely variable. When poison arrows were used on elephants, the shooter would have to track the animal for a day or two before the effect of the poison was sufficient to immobilize the animal.

## ANIMAL TOXICITIES

### Snake Envenomation

Reports of venomous snakebites are few in elephants. Envenomation is not usually a consideration in most captive elephants; however, venomous snakes may enter the grounds of zoos abutting woodlands or forests. Snakebite surely occurs in free-ranging animals, but making a diagnosis of venomous snakebite in an elephant is virtually impossible unless the strike is witnessed. Penetration of fangs into the subcutaneous tissue is likely possible only on the trunk or at the junction of the skin and the slipper of the foot.

Scott reports of an experience of a shikari (sportsman hunter) on a tiger hunt. The lead elephant was struck on the tip of the trunk by a king cobra *Ophiophagus hannah*. The 5443 kg (12,000 lb) elephant was dead in 3 hours.[35] The king cobra is the largest venomous snake in the world.

Scott also reports the death of a bull elephant working in the timber industry that was struck on the foot just above the nail by a king cobra and died. In Thailand, several elephants are reported killed every year by king cobras.[33,35]

Four types of venomous snakes are found in the United States: rattlesnake, *Crotalus* spp.; copperhead, *Agkistrodon contortrix*; water moccasin (cottonmouth), *Agkistrodon piscivorus*; and coral snake, *Micrurus* spp., and *Micruroides euryxanthus*; only large eastern *Crotalus adamanteus* or western *Crotalus atrox* diamondback rattlesnakes would pose a threat to an elephant. Table 33.6 lists some snakes that may pose a threat to elephants worldwide.

Clinical signs of rattlesnake bite include local tissue swelling at the bite site that spreads proximally and may involve any tissue or organ.[14] Systemic manifestations of envenomation are generally absent or minimal in large animals such as elephants. However, the bite from a large eastern diamondback rattlesnake, *Crotalus adamanteus*, could produce effects on the kidney and cardiovascular system (hypotension) as well as the local necrotizing effects.

The diagnosis may not always be clear if the bite was not observed, because trauma may produce similar signs. Supportive and symptomatic therapy may be instituted while monitoring the progress of the swelling. In some regions of the United States, malignant edema caused by *Clostridium septicum* may be a sequel to rattlesnake bite.

The only specific treatment for crotalid envenomation is the specific antivenin.[14] The amount of antivenin to be used is dependent on the amount of venom injected by the snake, not the size of the victim, and since that is not possible to determine, multiple vials (2 to 4), should be administered intravenously. Antivenin is prepared from horse serum and may sensitize the elephant to future use of any equine-prepared serum product. Seventy percent of airflow to the lungs is via the trunk. A snakebite near the tip of the trunk could occlude the nostrils and cause dyspnea.

In India and Southeast Asia the Russell's viper *Vipera russelli*, family Viperidae, and the king cobra *Ophiophagus hannah*, family Elapidae, are capable of envenomating an elephant.

In Africa consider the following: The black mamba *Dendroaspis polylepis*, family Elapidae, is found in East, South Central, and South Africa. The African puff adder *Bitis arietans*, family Viperidae, is found throughout much of Africa south of the Sahara Desert. The Gaboon viper *Bitis gabonica*, family Viperidae, is found in West Central and South Central Africa. The Gaboon viper is not only the largest viper, but it also has the longest fangs of any snake, 5 cm (2 in).

### Miscellaneous Animal Bites and Stings

Occasionally, all animals are stung or bitten by insects or arachnids (wasps, bees, ants, mosquitoes, and spiders).[14] Reactions from a single sting in large animals vary but are unlikely to produce serious illness. The local inflammatory response produced by venom injection cannot easily be differentiated from contusions or foreign body penetration (slivers).

Multiple bee or wasp stings may produce systemic manifestations. Horses and cattle have died from the envenomation when subjected to a swarm attack from a disturbed hive of bees or wasps. Africanized honeybees *Apis mellifera adamsonii* are now in the United States. They are no more toxic than European honeybees *Apis mellifera*, but are much more aggressive. Fire ants, *Solenopsis invicta*, found in the southeastern United States, have produced serious injury and even death to livestock tied near an ant hill and unable to flee. Neonates and moribund animals are especially at risk.

Only one report of an elephant death from insect envenomation was found, from a swarm of giant Indian bees *Apis dorsata*.[5,14] There have been no reports of spider or scorpion envenomation in elephants.

## DRUG TOXICITY

No drugs have been approved for use in elephants by the Federal Drug Administration (FDA) in the United States or similar agencies in other countries, and only a few have been tested for efficacy or safety in elephants. In the United States, extra-label usage of medications must be carried out according to the rules promulgated by the

**Table 33.6.** Venomous Snakes Potentially Lethal to Elephants

| Snake, Common/Scientific | Geographical Distribution | Size | Venom | | | Misc. Comments |
|---|---|---|---|---|---|---|
| | | | Mg of Dried Venom | Toxicity $LD_{50}$, Mouse IV mg/kg | Type | |
| **Asia** | | | | | | |
| King cobra/ *Ophiphagus hannah* | Throughout the range of the Asian elephant, India, Southeast Asia | 2.1–4.0 m (7–13 ft); max 5.58 m (12.3 ft) | | 1.6 | Neurotoxic | Oviparous, defends nest, eats other snakes |
| Russell viper/ *Vipera russelli* | India, eastern West Pakistan, Sri Lanka, southeastern China, Taiwan | 1.02–1.27, max 1.6 (2.25–2.8 ft) max 3.5 ft | 150–250 | 0.30 | Hematoxic, cytotoxic | Kills more people than any other snake worldwide |
| **Africa** | | | | | | |
| Egyptian cobra/ *Naja haje* | North Africa, Egypt, Sudan, East Africa, and south to northern Republic of South Africa | 1.2–2.4 (4–5.3 ft) | | 0.42 | Neurotoxic | Most widely distributed snake in Africa |
| Black mamba/ *Dendroaspis polylepis* | East Africa and south to northern Republic of South Africa | 2.7–4.3 (6– 9.5 ft) | | 0.25 | Neurotoxic | World's fastest snake, 20 kph (12 mph) |
| African puff adder/ *Bitis arietans* | Throughout much of sub-Saharan Africa | 0.9–1.5m (2–3.3 ft) | | 0.42–2.0 | Cytotoxic, necrotoxic | Causes most human snakebites in Africa |
| **Australia** | | | | | | |
| Common brown snake/*Pseudonaja textilis* | Eastern half of Australia | 1.2–1.8 m (2.6–6.0 ft) | 2 mg | 0.02 | Neurotoxic, hemolytic, cytotoxic | |
| Tiger snake/ *Notechis scutatus* | Southeast Australia, Tasmania, extreme southwest Australia | 1.0–1.8m (3.4–6 ft) | 35 mg | 0.26 | Neurotoxic | |
| **South America** | | | | | | |
| Neotropical rattlesnake/ *Crotalus durissus terrificus* | Brazil, Bolivia, Paraquay, Uruguay, Argentina, southern Mexico, and western Central America | 1.53 m (3.4 ft) | 24–44 | 0.13–0.35 | Cytotoxic, necrotoxic | |
| **North America** | | | | | | |
| Eastern diamondback rattlenake/ *Crotalus Adamanteus* | Southern Mississippi, Georgia, eastern North and South Carolina, Florida | 0.81– 2.5 m (1.75–5.5 ft) | 370–700 | 1.2–2.4 | Cytotoxic, necrotoxic | |
| Mojave rattlesnake/ *Crotalus scutulatus* | Southern California, Arizona, and central Mexico | 0.56–1.02 m (1.25–2.25 ft) | 50–90 | 0.21 | Neurotoxic, cytotoxic | Responsible for most human fatalities in California and Arizona |

FDA. Nonetheless, it is necessary to use these drugs in order to practice good medicine and surgery. That some risk may be involved in such use must be recognized. On balance, thousands of experiences indicate that elephants tolerate the use of drugs as well as other livestock species. Empirical dosages are given, usually based on size and consideration of doses used for horses. No unique drug idiosyncrasies have been reported in elephants, but drug toxicities have been seen, usually associated with an overdose.

## Tetramisole Toxicity

Tetramisole is an anthelmintic related to the more common levamisole. A 2-year-old Asian elephant was given

300 g of tetramisole orally.[21] If these figures are correct, and assuming this animal is close to 3 years old, the administered dose was 425 mg/kg, which is a massive overdose above the appropriate elephant dose of 2 to 4 mg/kg.

Clinical signs included muscle tremors, salivation, diarrhea followed by constipation, and bradycardia.

### Chloropromazine

Also known as thorazine or largactil, chloropromazine was a popular tranquilizer a few years ago, but is no longer available on a veterinary label in the United States. Chloropromazine is absorbed well when administered orally and as such was used to sedate a bull Asian elephant that became fearful of crowds (ochlophobia) while being used in prolonged festival ceremonies.[8] Two thousand mg administered orally twice daily quieted the bull, but also made him drowsy.

In another instance, however, a single dose of chlorpromazine given orally caused the death of an elephant.[30] Furthermore, if chlorpromazine is inadvertently administered intraarterially it causes an endarteritis and thrombosis of terminal arterioles, resulting in necrosis of the tissue supplied by that vascular bed. This would be a factor if chlorpromazine was administered intravenously, because it is not uncommon for an artery to be cannulated instead of a vein.

### Atropine

Atropine is a parasympatholytic agent that is frequently used in anesthesia to increase heart rate and decrease respiratory and gastrointestinal secretions. In large animals it is a potent inhibitor of gastrointestinal motility. It is also used to treat organophosphate insecticide toxicity. Atropinelike alkaloids (scopolamine, hyoscine) are present in certain plants in the family Solonaceae (*Atropa belladona, Helloborus niger, and Datura* spp.). Scopolamine and hyoscine have been included in drug mixtures for immobilization of elephants. However, scopolomine tends to lengthen recovery and is no longer considered appropriate.

**Signs.** Atropine toxicity is characterized by an exaggeration of the pharmacologic action, including mydriasis, dry mucous membranes, and gastrointestinal atony. At high toxic doses atropine affects the brain, causing central nervous system stimulation including convulsions and maniacal behavior. An elephant experienced a period of excitement (swaying, kicking, nonresponsiveness to commands, and moving around the stall in an agitated manner) following the intravenous administration of atropine at a dose of 0.05 mg/kg.[23] A more appropriate IV dose would be closer to 0.01 mg/kg.

**Diagnosis.** Diagnosis includes a history of administration of atropine and clinical signs.

**Management.** Excitement and mania should be controlled by sedation. Avoid the use of phenothiazine tranquilizing agents because they may cause exacerbation of convulsions. Diazepam may be used. In small animals and horses, barbiturates would be used for sedation. However, in elephants, access to a vein may be difficult, so it may be necessary to sedate with an immobilizing agent such as medetomidine or xylazine. It would seem logical to counteract the effects of atropine with parasympathomimetic agents (pilocarpine or physostigmine). Although these drugs may counteract the peripheral effects, only central nervous system depressants are effective in alleviating peracute atropine toxicity.

### Perivascular Administration

Phenylbutazone (butazolidin) is highly irritating if accidentally administered perivascularly or if there is leakage around a needle during intravenous administration. Segmental necrosis and sloughing of the pinna may result.[28] Other drugs that cause phlebitis of elephants are diclophenac (Cataflam®) and thiopentone.[8] Trimethoprim-sulfa (Tribrissen®) was administered intravenously in an elephant with salmonellosis. Forty-eight hours after the last administration, a segment of the pinna began to necrose where some of the medication had apparently been deposited extravascularly.[41]

### Acepromazine

Acepromazine was suspected of causing photosensitization in five elephants.[8] Acepromazine is a derivative of phenothiazine, a known photodynamic agent, but there are no reports of photosensitization from acepromazine in other domestic or wild animals.

## INTENTIONAL POISONING

### Suxemethonium

Culling of elephant herds in South Africa employed the use of suxamethonium to paralyze elephants, which was followed by shooting. Suxamethonium at a dose of 1 g intramuscularly has also been used to euthanize elephants.

### Lysergic Acid Diethylamide (LSD)

Lysergic acid is a constituent of ergot alkaloids. LSD is a synthetic product that produces hallucinations in people and was used in the 1950s and 1960s in the drug culture.

LSD was administered to a 3175 kg bull elephant with the purported objective of producing a syndrome similar to musth.[43] The drug was administered intramuscularly at a dose of 0.094 mg/kg (297 mg). This was an exorbitantly high dose; the dose for a 100 kg human is 0.003 mg/kg. This was totally inappropriate research and resulted in the death of a fine young bull elephant. Supposedly this experiment was duplicated without the

death of the elephants involved,[37] but again, it was needless research.

Five minutes following administration the elephant trumpeted loudly, collapsed, and began convulsing. The elephant died 1.5 h later in spite of medications given to try to counteract the effects of the LSD.

### Zinc Phosphide ($Zn_3P_2$)

Zinc phosphide is a potent rodenticide usually placed in a bait such as bread, bran mash, or soaked grains and sugar. Zinc phosphide may cause mortality in a broad host range, including elephants.

The mechanism of action is the liberation of phosphine gas ($PH_3$) when exposed to hydrochloric acid and water in the stomach. The bait plus other food in the stomach enhances the release of phosphine.

Two elephants were apparently intentionally poisoned with zinc phosphide in an Indian Zoo (news release, October 2004). Both animals had an acute hemorrhagic diarrhea and dyspnea. Phosphine gas is a powerful irritant and produces a hemorrhagic gastroenteritis and endocarditis. Zinc phosphide was identified in gastrointestinal contents. More selective rodenticides are usually used in the United States, but zinc phosphide is readily available in the city where the zoo is located.

## ELEPHANT EUTHANASIA

It is always emotionally traumatic to all concerned when it becomes necessary to end the life of any animal—especially an elephant, because trainers, keepers, handlers, veterinarians, and the public become attached to elephants in a special way. Those responsible for carrying out euthanasia must appreciate the associated emotions and act appropriately.

Many different methods have been used, including shooting, incising the aorta with a scalpel in a hand inserted deep into the rectum, use of euthanizing agents used for domestic animals, and an overdose of an immobilizing agent.

Shooting, except by a professional, experienced hunter, with an appropriate weapon, is fraught with possible incomplete immobilization, and it is not recommended. Incision of the aorta requires that the elephant tolerate a rectal examination. However, the procedure is without pain and internal hemorrhage will quickly bring about anoxia and death. This may be appropriate for a moribund elephant.

An overdose of euthanizing agents (toxic barbiturates) used for companion animals and livestock requires intravenous injection because there is no pleural space in elephants and intraperitoneal infusion is not an option. Additionally, the cost may be prohibitive for an animal the size of an elephant.

In a day when immobilizing agents are available in all but the most remote locations on the globe, the humane method of euthanasia in the elephant is to first immobilize with an agent that renders the elephant unconscious (etorphine, carfentanil, Xylazine, detomidine). A catheter is placed in an accessible vein on the caudal aspect of the ear or the saphenous vein on the medial side of the tibia. Place the catheter quickly following recumbency and incise down to the vein if blood pressure drops or there is a problem with getting the catheter through the skin. Then a saturated solution of potassium chloride (KCl) is injected intravenously. In a few seconds the heart will stop and death is instantaneous.

Powdered KCl is readily available from chemical companies or a pharmacy without a prescription being necessary. It need not be medical grade, and sterility is not necessary. A saturated solution is prepared by adding 180 g of KCl to 600 ml of hot water, which provides 0.3 g/ml of solution. The dose required to stop the heart is 44 mg/kg (20 mg/lb). For a 3000 kg elephant it would require 440 ml of the 0.3 g/ml saturated solution. In practice, the solution is administered in excess, so it would be appropriate to make a double quantity of KCl solution to account for possible spillage. This method of euthanasia is humane and suitable for any large domestic or wild animal.

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# 34 Zoonoses and Human Injury

Joel Maslow

## INTRODUCTION

Elephants are at risk for and succumb to a number of infectious diseases. Many of the same diseases affect humans with a subset considered as communicable and a smaller subset as true zoonoses. The first part of this chapter reviews the infectious diseases of elephants with respect to their zoonotic potential. For detailed descriptions of disease presentations, see Chapter 11. The second half of the chapter reviews the topic of human injuries and fatalities resulting from direct contact with elephants. Incidents cited in the literature and available over the Internet were collated. Events from countries with endemic elephant populations are discussed separately from countries with captive elephant populations. If possible, fatalities occurring within the United States (U.S.) were verified from primary documents or by interview. Statistics outside of the U.S. are listed without verification. The intent of this portion of the chapter is to present an overview to assess potential risk factors for injury and death with the goal to minimize morbidity through increased preparedness.

## INFECTIONS OF ELEPHANTS AND THEIR ZOONOTIC POTENTIAL

Table 34.1 lists diseases of elephants with respect to their zoonotic potential. References are provided for those diseases with documented interspecies transmission.

### Bacterial Diseases

**Anthrax.** Human infection with anthrax typically results from contact with infected animals or animal products. Occasional cases in the U.S. are considered to result from contact with spores surviving in soil. Syndromes include cutaneous disease that presents as a nontender papule with surrounding edema that proceeds to form a central eschar; inhalational anthrax that presents with nonspecific flulike symptoms with subsequent hypoxia and dyspnea; and gastrointestinal anthrax that may cause mucosal necrosis and gastrointestinal bleeding.[11] All forms are fatal if untreated.

Anthrax is prevalent among wild-animal populations, especially during the dry season (see Chapter 11 for more details). A single case of human anthrax was linked to elephant tusks used in the piano industry.[39] A factory worker involved in making ivory for piano keys presented with a finger ulcer at the site of minor trauma and rapidly developed sepsis and died. Blood cultures yielded *B. anthracis*.

**Colibacillosis.** Zoonotic infection of enteropathogenic forms of *Escherichia coli*, such as with serogroup O157:H7, occur as a result of contact with colonized cattle. Although *E. coli* is considered a pathogen for elephants,[36] zoonotic spread has not been documented.

***Mycobacterium avium.*** In humans, *Mycobacterium avium* presents primarily as pulmonary or disseminated disease. Pulmonary infection is indistinguishable from tuberculosis (TB) caused by *Mycobacterium tuberculosis,* is typified by apical cavitation, and occurs primarily in those with chronic obstructive lung disease. Disseminated infection occurs mainly in immunosuppressed patients, particularly those with end-stage AIDS. *M. avium* is the most prevalent mycobacterial species cultured from elephants[31] although this has unclear clinical significance.[28] Interanimal transmission of *M. avium* has been described for immunosuppressed, SIV-infected macaques that ingested contaminated feces from common waste troughs.[21] Conversely, exposure to contaminated potable or natural water sources has been a source of infection for animals as well as humans.[43,48] To date, *M. avium* has not been definitively documented as a cause of clinical disease in elephants, and there is no data supporting zoonotic transmission of *M. avium* from elephants or from other animals—nor would this be considered likely.

**Table 34.1.** Zoonotic Potential for Infections of Elephants

| Infection | Group* | References of Zoonotic Disease |
|---|---|---|
| Bacteria | | |
| *Bacillus anthracis* | I | Seideman 1947 |
| *Escherichia coli* | III | |
| *Mycobacterium avium* | III | |
| *Mycobacterium bovis* | II | |
| *Mycobacterium elephantis* | III | Shojaei 2000, Turenne 2002 |
| *Mycobacterium tuberculosis* | I | Michalak 1998, Oh 2002, Lewerin 2005 |
| *Pasteurella multocida* | III | |
| *Salmonella* | II | |
| *Yersinia pestis* | III | |
| Viruses | | |
| African horse sickness virus | III | van der Meyden 1991 |
| Foot and mouth disease | III | Mayer 1973 |
| *Orthopoxvirus bovis* | I | |
| Rabies | II | |
| **Protozoa** | | |
| *Cryptosporidium* | II | |
| *Toxoplasma gondii* | III | |

*Group Definitions: I—Definitive evidence of interspecies transmission between humans and elephants; II—Infections considered to be at high risk for interspecies transfer; III—Infections considered to be at low risk for interspecies transfer.

***Mycobacterium elephantis.*** *M. elephantis* is a newly described species genetically related to *Mycobacterium confluentis* and *Mycobacterium smegmatis* that was originally cultured from the lung of an elephant with chronic respiratory disease.[41] *M. elephantis* has been identified in cultures from 8 Canadians and 3 Canadian immigrants, including 10 with pulmonary infection and 1 with lymphadenitis.[45] Zoonotic spread was not considered as likely by the authors nor would it be considered as likely.

***Mycobacterium tuberculosis* complex.** Estimates by the World Health Organization are that almost one-third of the world's population is infected with *M. tuberculosis,* causing over 3 million deaths yearly. Pulmonary disease is most common, with lymphoreticular disease of the spleen, liver, and lymph nodes as second most common. The public health risk of TB directly relates to the level of bacterial shedding into the sputum and exposure to contaminated aerosols.

*M. tuberculosis* is the second most prevalent mycobacterial species to infect elephants.[26,29] Reports of TB prior to 1996 were uncommon but not rare. Elephant-to-elephant transmission of *M. tuberculosis* has been demonstrated for three cohorts of animals.[19,28,49] Transmission between elephants and other zoo animals has also been described.[19,30,49] *M. tuberculosis* has also been transmitted between elephants and humans and was confirmed by genetic analysis of infecting isolates.[25] Additionally, in the latter outbreak, public health investigation found that 11 of 22 employees demonstrated intradermal reactivity to purified protein derivative (PPD) including 3 that had skin-test conversions during the time of follow-up; i.e., had evidence of recent infection.[25] Because prior skin test data for staff and cultures for elephants were not available, it could not be determined whether the index case was an elephant or human.[25] A separate study examined 307 employees at the Los Angeles Zoo after *M. tuberculosis* was diagnosed in elephants, goats, and a rhinoceros.[30] Almost 20% of the staff had positive skin-test reactions, although, as above, prior skin-test results were not reported. Univariate risks associated with a PPD-reactivity included male gender, work as a groundskeeper or construction worker, and attendance at an elephant necropsy. Finally, a report by Greenberg alleged that an animal trainer with cavitary tuberculosis was the source of a fatal infection for a circus elephant;[10] however, the infecting isolates were of different phage types and thus were distinct strains.[12]

To effectively prevent transmission of *M. tuberculosis* from elephants (or other animals) requires an understanding of the pathogenesis of disease, the diagnosis of infection, and infection control practices that have been proven as effective. To reduce the public health risk of TB from elephants, suspect animals should be placed into quarantine at distances sufficient to prevent transmission of infected droplets. Personal protective equipment should be worn around suspect animals, including gowns, gloves, masks (N95 respirators), hair covering, and eye protection. Trunk wash represents the current gold standard for diagnosis.[26,27] Other methods have yielded conflicting results,[26] although serology is promising[18] (see Chapter 11 for more details).

Similar to man, *M. tuberculosis* infection in elephants is primarily pulmonary,[28] although disseminated disease may occur affecting the spleen, bone marrow, and adrenal glands. Public health risk of *M. tuberculosis* and *M. bovis* relates primarily to the degree that tubercle bacilli are shed into the sputum or other body fluids. Animals without active shedding do not pose a risk for transmission. Coughing, trumpeting, and other vocalizations produce aerosols that can be inhaled. Although less common, significant exudative disease of the vaginal tract could aerosolize and be inhaled or ingested. Transmission risk can also be reduced by treatment of infected animals to reduce shedding.[28] Treatment guidelines for elephants are based on recommendations for humans and were published in 2003.[46]

**Mycobacteria other than tuberculosis (MOTT).** MOTT are infrequently cultured from trunk wash specimens and have unknown disease potential.[28,31] Their zoonotic potential is considered negligible akin to *M. avium*.

***Salmonella.*** Nontyphoidal strains of *Salmonella* are a common cause of diarrheal disease in humans and occasionally cause septicemia. *Salmonella* may also cause di-

arrhea and septicemia in both African and Asian elephants.[23,53] There have been no confirmed cases of interspecies transmission of *Salmonella* to or from elephants. One report without microbiologic confirmation postulated that human refuse may have been the source of infection for a juvenile elephant that died of *Salmonella*.[23] The risk of possible zoonotic infection should be considered high for infected animals, although the expected prevalence in elephants and other herbivores is considered low.

**Streptococcosis.** Hemolytic streptococcal infections in humans include skin and soft tissue infections, bacteremia, pneumonia, pharyngitis, and endocarditis. One paper reports on ulcerative infection of the soles of the feet of individuals walking barefoot in a region where a domesticated elephant was tethered.[34] Although zoonotic infection was suggested, the organism was not isolated from the elephant or from elephant feces.

***Yersinia pestis* (Plague).** Human infection with the plague bacillus, *Yersinia pestis* results from infected rodents via an arthropod vector. Pneumonic plague is a rapidly progressive, fatal pneumonia that is highly communicable. Bubonic plague is characterized by enlarged draining lymph nodes (buboes) and results in sepsis and death if not treated. Antibodies to *Y. pestis* have been documented in 0.3% of South African elephant sera.[8] Disease in elephants has not been described and zoonotic transmission would not be expected.

### Viral Diseases

**African horsesickness virus.** Serologic evidence of infection with African horsesickness virus (AHSV) has been documented in elephants, typically with low complement fixation titers. Moreover, Culicoides midges, the arthropod vector of AHSV, have been isolated from retroauricular areas of African elephants and utilize elephant dung for reproduction and growth.[24] Human infection with AHSV has occurred in four laboratory workers involved in vaccine production; the infection was characterized by fever, headache, encephalitis, mental slowing, confusion, and short-term memory loss and chorioretinitis along with retinal hemorrhage.[35,47] There is, however, no suggestion that the disease is zoonotic from elephants or other animals.

**Poxvirus.** Poxvirus infection in elephants occurs as an epizootic infection involving *Orthopoxvirus bovis*, or cowpox, spread via rodent vectors. Elephants present with a vesicular skin eruption that may progress to generalized ulcers.[15] Congenital disease has also been reported following vaccination of a pregnant elephant cow.[64] Zoonotic transmission of poxvirus infection from infected elephants was reported for two zoo workers who assisted at the necropsy of elephants at the Munich Zoo.[22] Both workers developed vesicular lesions on the upper extremities along with fevers and healed with scarring.

**Rabies.** Human rabies infection is a zoonotic illness resulting from exposure to infected animals, typically bats, raccoons, dogs, and foxes. The organism is shed into the saliva of affected animals and can cause infection through puncture wounds incurred by bites. Elephants present with a progressive neurologic illness marked by aggressive behavior, anorexia, difficulty in standing followed by paralysis, and death.[52] Although elephant-to-human transmission of rabies has not been described, the risk of transmission from a significant mucous membrane exposure of saliva from an infected elephant is unknown but theoretically possible.

### Fungal Disease and Protozoa

None of the fungal or protozoal diseases of elephants are considered to be potentially zoonotic.

**Toxoplasmosis.** Serologic evidence of *Toxoplasma gondii* infection has been demonstrated in 45% of captive Thai elephants in one study without mention of disease.[44] Because elephants are not definitive hosts for toxoplasmosis, elephant-to-human transmission is not considered possible.

**Cryptosporidiosis.** *Cryptosporidium* spp. infection is a common cause of human diarrheal disease in both normal and immunosuppressed hosts. *Cryptosporidium* was shown to colonize an African elephant persistently at the Barcelona Zoo[9] and thus could serve as a source for human exposure.

## INJURY AND DEATH RELATED TO HUMAN-ELEPHANT INTERACTIONS

Contact between elephants and humans has been lengthy and storied, dating to the Stone Age when hunting of mastodons, documented in cave drawings, proved fatal for hunted and hunter. Elephants were used in executions in Asia 4,000 years ago to crush the heads and chests of condemned individuals.[51] Although the intersection between human and elephant habitats in countries with endemic populations has resulted in the largest number of deaths and injuries worldwide, incidents involving captive animals garner the most publicity.

### Elephants in Captivity

A compilation of reported fatalities and injuries directly attributable to contact between humans and captive elephants in countries without endemic elephant populations is listed in Table 34.2. Unfortunately, press reports[3] include more sensational aspects of incidents and have not reported corrections to original stories. Additionally, compilation of events reported by certain other

**Table 34.2.** Injuries and Fatalities Reported as a Result of Direct Contact Between Elephants and Humans

| | | United States | | International | |
|---|---|---|---|---|---|
| Year | Locale | Fatalities | Injuries | Fatalities | Injuries |
| 2000–2005 | Zoo | 1 | 3 | 4 | 4 |
| | Circus | 2 | 1 | 1 | 0 |
| | Other* | 0 | 1 | 8 | 2 |
| 1996–1999 | Zoo | 1 | 4 | 3 | 0 |
| | Circus | 0 | 5 | 4 | 0 |
| | Other | 0 | 2 | 3 | 4 |
| 1990–1995 | Zoo | 4 | 6 | 5 | 1 |
| | Circus | 4 | 9 | 4 | 1 |
| | Other | 0 | 6 | 4 | 5 |
| Pre–1990 | Circus | 3 | 0 | 0 | 0 |

*Other facilities include wildlife parks and safaris, ride and show animals.

Data were compiled from reported sources (Langley 2001, Shellabarger 2001, Scigliano 2002, Albrecht 2003, API 2005, Associated Press 2005, PETA 2005, Schwammer 2005). As possible, data were corroborated from primary print sources or by contacting the affected facility (for U.S.–related events). Reports of deaths appeared to be accurate; however, details surrounding the events as reported from primary source material differed many times from that listed in summary lists.

sources[2,13,32,38] appear only to cite original journal sources without verification of details. A total of 15 U.S. fatalities have been reported since 1990. The number of fatalities and injuries has dramatically decreased over the past 10 years, with incidents involving circus animals showing the greatest decline, and that may be the result of education and better training (personal communication, H. Harriet). Although deaths and serious injuries involving U.S. zoos and circuses in general involve a single event at each facility, exceptions are noted.[2,32,38] The majority of incidents involving zoo, circus, and other commercial venues (e.g., game parks, safari parks, public rides, and animals used for public showings) appear to be the result of accidents. For example, one recent event was reported as a trampling of an animal keeper,[3] whereas the coroner's report ascribed death to accidental trampling after the keeper was rendered unconscious from slipping and hitting his head while exiting the elephant area (personal communication, L. Solheim).

Unfortunately the decrease in elephant-related fatalities observed in the United States has not been observed for captive animals elsewhere in the world (Table 34.2). There were 10 deaths reported from European countries and 6 from the Americas other than the United States. The greatest number of deaths reported was for England with 4 fatalities, 3 since 2000, of which 3 involved animal handlers in zoos. The ratio of injury to death was 1:2 worldwide compared to 2:1 in the United States, possibly a reflection of lower reporting.

## Countries with Endemic Populations of Elephants

As expected, countries with endemic populations of elephants report significantly higher numbers of incidents[6] (see also www.savetheelephants.org, www.elephantcare.org, and www.circuses.com/attacks-ele95.asp). Most incidents and figures are unconfirmed. Because some events are reported by multiple news services, overestimation may occur if citations are counted separately without careful inspection. As above, reports of deaths are cited in compilations reported on the Internet[32] with no indication of verification.

Of Asian countries with an endemic elephant population, India has the largest number of reported deaths with 19 per year documented in two provinces between 1980 and 1991[6] and an average of ~175 deaths yearly countrywide from 1991–2001.[5] One article from Bangladesh refers to a report from The World Conservation Union that cites 162 elephant-related deaths (~27 per annum) and 348 injuries between 1997 and 2002.[13] Similarly, a posting quotes a figure of 60 deaths yearly in Sri Lanka.[50] Other countries with reported deaths ascribed to elephants include Cambodia, Indonesia, Malaysia, Nepal, Singapore, Thailand, and Vietnam (www.savetheelephants.org). The vast majority of incidents relate to the intersection of human and elephant habitats, with a minority involving privately owned animals.

On the African continent, Kenya and South Africa have the greatest number of reported deaths (www.savetheelephants.org). Deaths in Kenya from 1989 through 1996 have been quoted to range from 8–37 per year with a peak between 1991–1993[14] (see also www.elephantcare.org). Other countries with elephant-related reports of deaths include Botswana, Ethiopia, Kenya, Malawi, Namibia, South Africa, and Zambia. South Africa appears unique in that many deaths involve tourists or rangers working in game parks.[7,16]

## Risk Factors for Injury from Elephants and Risk Reduction

Identification of risks for injury or death to humans in zoos, circuses, or other commercial venues is difficult because few reports provide detailed objective information. Reports suggest that accidents such as falls in an

enclosed space housing one or more elephants may be one of the more frequent causes of injury or death by trampling. Alcohol intoxication was likely a factor in a few deaths. Feeding of elephants by those unfamiliar to the elephants has been associated with deaths, especially children. Loud noises or other inciting factors have caused animals to startle, resulting in death or injury. Finally, some deaths appear to have occurred after elephants were provoked.

In endemic areas, crop raiding, salt seeking, and encounters during periods of elephant migration appear to result in many incidents.[33,42] Raiding by elephants was more common at night in two studies from Kenya and India.[6,42] Although male elephants were most frequently associated with injuries and deaths, subadult male elephants were more likely to attack. Alcohol intoxication by humans as well as ingestion of fermented rice by elephants may have been a factor in deaths in Asia[6] (see also www.savetheelephants.org). Illness in elephants and periods of musth may increase the chance of attack, as noted in a study of tourists visiting game parks in South Africa.[7]

Two studies have promoted the use of geographic localization of elephant migration patterns using geographic information system (GIS) or global positioning system (GPS) technology to predict interaction points and to serve as an early warning system.[33,42] Other remedies to reduce elephant raiding have included spreading chilies or chili oil around villages, dissemination of tiger urine at the borders of villages, electrified fencing, and relocation of problem animals (www.savetheelephants.org).

### Human Injuries

Detailed descriptions of human injuries are lacking as a rule; however, some reports[1,2,3,6,7,16,32,37,38,40,51] (see also www.savetheelephants.org) allow the classification of elephant-related injuries into three broad groups: crush injuries, puncture wounds, and falls.

Crush injuries are the most common and may occur from trampling, squeezing by an elephant's trunk, or compression between an elephant and other solid object. These may involve any body part, but the most serious are those to the head, neck, and thorax, which are also the most common areas affected.

Puncture injuries to the thorax and abdomen are next most common and may cause trauma to vital organs, including the heart, lung, liver, spleen, and viscera, or cause damage to major blood vessels. Finally, falls from heights (whether accidental or intentional) have resulted in fractures, including limbs, skull, pelvis, and vertebrae. Any fall should also raise suspicion for possible cervical spine injury.

First aid preparedness may help reduce the morbidity and mortality following injury. Training is crucial to maximize success. Persons with known or suspected head or neck trauma should not be moved until the neck and cervical spine have been immobilized by trained first aid providers. Temporizing measures include placement of objects such as sand bags or rolled towels around the head, neck, and body to limit movement and maintenance of body temperature in case of shock. Chest and abdominal trauma may result in respiratory difficulty or blood loss from puncture of vital organs or other vital structures and would benefit from oxygen treatment. In the event of cardiac or respiratory arrest, cardiopulmonary resuscitation (CPR) should be initiated. Injuries that are not as usual, such as traumatic amputation or partial transection of a limb, should be addressed immediately by placement of a tourniquet above the point of injury to prevent significant blood loss. In summary, it is recommended that individuals skilled in basic first aid including CPR be present on site at facilities. Oxygen, devices (sand bags, towels) to limit movement of the head and neck, and tourniquets should be available. Use of tourniquets should be limited to trained staff or to times of true emergency.

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# 35 Veterinary Problems of Geographical Concern

## Introduction

Susan K. Mikota and Murray E. Fowler

Many of the health problems of elephants in captivity are shared across regions, but there are also geographically unique problems. The intent of this chapter is to present an overview of these similarities and differences. The information that follows reflects both published data and the firsthand experience of veterinarians who work with elephants in range countries.

Veterinarians from Asia and Africa were invited to participate. Contributions were received from 10 of the 13 Asian range countries. Unfortunately China, Bhutan, and Thailand are not represented.

Authors were asked to inform readers of the status of free-ranging and captive elephants and to describe common and unusual problems, therapeutic methods, and available veterinary products. In response, they provided a wealth of information. It was impossible to include it all. The editors have attempted to provide an overview of regional problems without repeating the details (diagnosis, clinical signs, epizootiology, etc.) of specific infectious and parasitic diseases. These are discussed in other chapters. Rather, the editors have strived to include interesting information not mentioned elsewhere—an unusual condition, a detailed treatment protocol, or a traditional remedy.

Valuable and practical elephant health care information often remains locked in the unpublished experiences of veterinarians. This anecodotal information has been and will continue to be an important resource.

This chapter provides only a brief insight into elephant medicine in Asia and Africa. It is hoped, however, that it will inspire the greater global sharing of information among veterinarians who care for these remarkable animals.

Elephant veterinarians are encouraged to continue sharing information via the website maintained by the editor (Mikota) at Elephant Care International: www.elephantcare.org.

## Section I—Africa

Jacobus G. du Toit

### INTRODUCTION

In most African countries elephants are found in National Parks or on government land. In South Africa, Namibia, and Zimbabwe, elephants are also found on privately owned game reserves. Between 1979–2001 over 800 elephants were introduced to over 58 game reserves in South Africa.[7] The economic value of elephants on reserves makes veterinary care important. The role of the veterinarian in the management of the African elephant is largely focused on capture and translocation operations. Veterinary intervention with elephants in National Parks is primarily limited to injuries caused by man or to vaccination (e.g., for anthrax) programs to halt disease outbreaks in wild populations.

## DISEASES AND DISORDERS OF FREE-RANGING ELEPHANTS

### Infectious Diseases

See also Chapter 11 for more details.

#### Viral.

Herpesvirus (Cutaneous papillomatosis). Herpesvirus causes superficial pink-colored skin lesions (10–30 mm in diameter) that occur predominantly on the trunk and head and around the genital opening. The lesions are benign and disappear spontaneously; however, secondary infection may cause abscessation. Herpesvirus-associated intranuclear inclusion bodies have also been observed in pulmonary nodules in culled elephants.[12] There is no vaccine available.

Encephalomyocarditis virus (EMC). EMC occurred in free-ranging adult African elephant bulls in the Kruger National Park in 1993–1994.[8] During this period, 53 of 64 (83%) bulls died of cardiac failure. Postmortem lesions included hydrothorax, hydropericardium, severe ascites (up to 50 liters), and petechial and ecchymotic hemorrhages on the epicardium. There is evidence that rodents *(Otomys natalensis* and *Paraxerus cepapi)* carry the virus and spread it by urinating on food. Rodent control in the food storage facilities is therefore of the utmost importance for prevention. Onderstepoort Biological Products can manufacture an excellent vaccine on request (Onderstepoort Biological Products, Private Bag X7, Onderstepoort 0110, Tel: 27-12-522 1500).

#### Bacterial.

Anthrax. Anthrax may be spread by blowflies (*Chrysomya albiceps* and *Chrysomya marginalis*) that feed on infected carcasses and then vomit spores onto vegetation at a height of 1–2 meters above ground. Elephants become infected by ingesting contaminated food or water. Anthrax may also be spread mechanically by elephants moving between waterholes or by vultures that have fed on infected carcasses (see Figure 35.1). Elephant mortalities during outbreaks of anthrax have been low, varying from 0.28% in the Kruger National Park to 1.6% in the Etosha National Park.[4] A vaccine registered for cattle from Onderstepoort Biological Products has been used on private game reserves in southern Africa during outbreaks. Ill animals may be treated with penicillin. (PeniLA®, Virbac SA, P/Bag X 155, Halfway House 1685, Tel: 27-12-657 6000. The dosage for drugs used in elephants may be found in Chapter 15.

Tuberculosis (TB). TB has not been reported in wild African elephants. *Mycobacterium bovis* occurs in a wide range of wildlife species in places like Kruger National Park, however, so spillover into elephant populations is a concern.

Ulcerative pododermatitis. Thirteen adult bulls suffering from ulcerative pododermatitis were recorded over a 30-month period in the Kruger National Park. Distortion and overgrowth of the toenails was accompanied by a

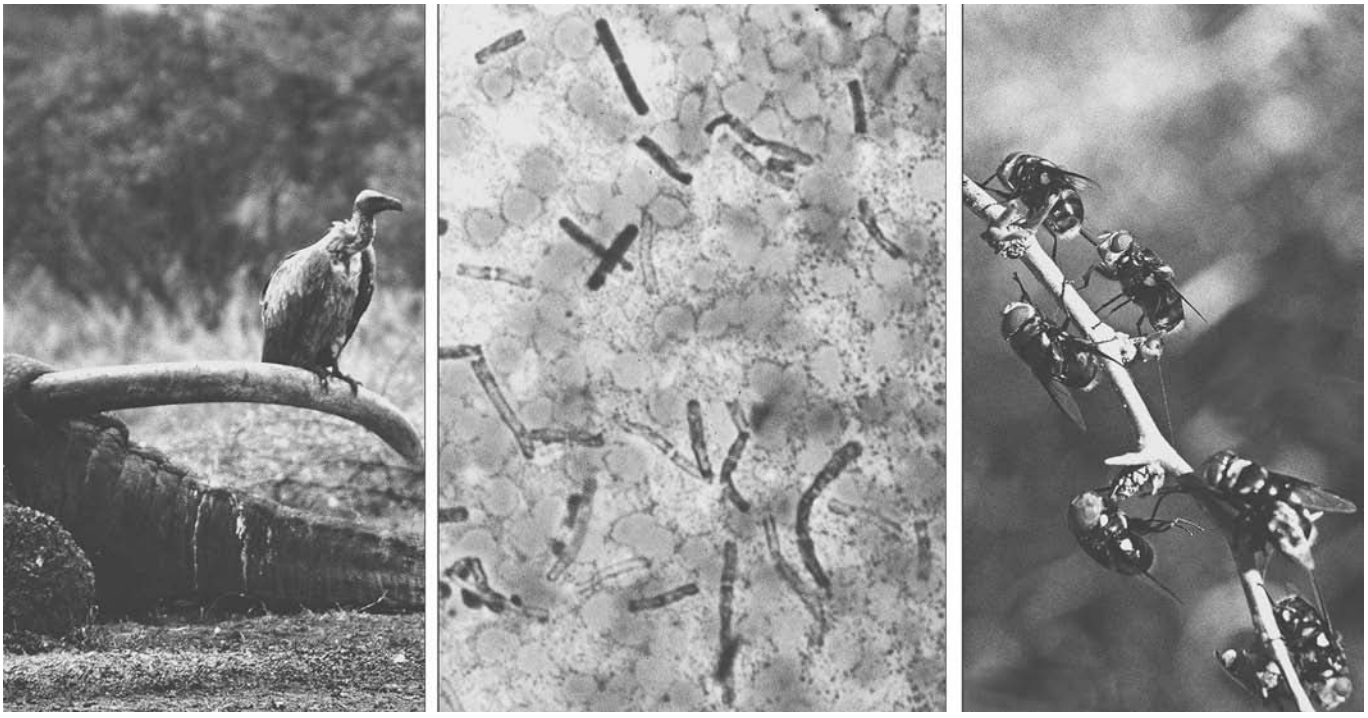

***Figure 35.1.*** *Anthrax is transmitted by mechanical vectors, such as blowflies and vultures, that contaminate browse and water with spores. Large cells with square ends and centrally located ellipsoid spores are characteristic of the anthrax bacilli (photograph of blowflies and vulture by D. F. Keet).*

***Figure 35.2.*** *Severe pododermatitis in a free-ranging African elephant (photograph by D. F. Keet).*

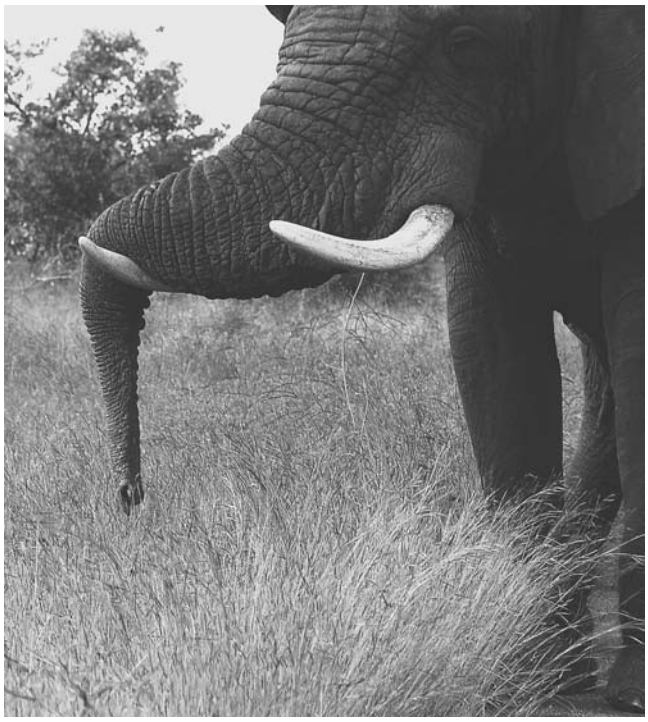

***Figure 35.3.*** *Floppy trunk syndrome in an African bull (photograph by D. F. Keet).*

septic vasculitis. S*treptococcus agalactiae* and *Dichelobacter nodosus* were the most consistently isolated organisms. The problem occurred during a severe drought and was thought to be due to penetrating injuries from dry wooden stumps[10] (see Figure 35.2).

**Tusk conditions.** Tusk conditions have been well described.[16] Only a few conditions have been noticed in free-ranging elephants.

Tusk sulcus infection. Traumatic impact when elephants push against solid objects may cause a purulent infection of the tusk sulcus. Healing usually takes place when the animals calm down in the bomas.

Blind tusks. Failure of the incisor to erupt through the skin may cause subcutaneous swelling. The elephant will push with its head against objects and show signs of discomfort. A surgical incision may be made through the skin. A gauze sponge dipped in a topical wound ointment may be placed as a plug to prevent myiasis.

Tusk pulpitis. Fractured tusks with exposed root canals may form fistulous tracts with purulent exudates. The fistula should be flushed twice daily with a 1:10 dilution of povidone-iodine (Betadine®, Astra-Zenica, Private Bag X 30, Sunninghill 2157, Tel: 27-11-797 6000). An antibiogram (culture and sensitivity) is important for the successful treatment of the wound. Bone cement is applied after the infection is resolved.

## Parasitic Diseases

The bile duct hookworm (*Grammocephalus calthratus*) may cause morbidity and mortality in newly captured elephants. Injectable levamisole (Tramisol®, Coopers, 11 Gibbon Rd., Baulkham Hills, New South Wales 2153, Tel: 02-9852 7200) is given routinely during capture operations. Spirurid worms (*Parabronema rhodesiense*) cause damage to the mucosa of the stomach and with associated stress will cause ulcers. The parasite may be treated with anthelmintics such as ivermectin (Ivomec®, Merial SA, PO Box 5924, Halfway House 1685, Tel: 27-11-315 8001).

## Noninfectious Disorders

**Floppy trunk.** Paralysis of the trunk has been observed in elephants in the Satara area of the Kruger National Park and Fothergill Island in Lake Kariba[11] (see Figure 35.3). The condition developed slowly over a period of months. The primary lesions occurred in the nervous system, and the muscle atrophy appeared to be secondary. The affected elephants adopted new techniques for feeding and drinking by using a front foot in conjunction with the trunk to scoop food and taking water directly into their mouths. Research is ongoing to determine whether plant toxins or heavy metals are causing this syndrome. The three most likely plant genera, as assessed by comparing FTS-affected and unaffected areas, are *Heliotropium*, *Indigofera*, and *Boerhavia*.

**Poisoning.** Elephants of the Hwange National Park in Zimbabwe died when they drank from dipping tanks containing organo-phosphates on cattle ranches.[4] This

may be a factor where wildlife sanctuaries with poor water sources are close to cattle ranches.

**Trauma.** Superficial wounds of the trunk and legs occur commonly among young animals from fighting. Abscesses may result and radial paralysis in a young wild elephant has been observed.[9] "Floppy ear" is a condition that results when an elephant gets its head wedged in a tight area and must struggle to free it. The cartilage tears but the skin stays intact. The damage is permanent and these animals are especially prone to sunburn. Young elephants can panic easily and may run into veldt fires in the absence of a matriarch, resulting in severe burns. Breaking of tusks occurs when newly caught elephants charge people during the boma-training period.[5] Land mines, snares, and poaching injures or kills elephants. Such human-inflicted injuries are the most frequent cause of trauma and death in elephants on the African continent.

**Subcutaneous edema.** Edema occurs with anemia, liver fluke infection, or conditions that lead to hypoproteinemia. Ventral edema usually manifests overnight. A theory that the cause is due to stress is questionable. Gravitation of fluids to the umbilical area may lead to pressure necrosis and sloughing. Ventral edema has been treated successfully with hot and cold pressure bandages.[5]

**Iatrogenic.** A condition known as "pink foam syndrome" has been described in elephants that died during capture operations.[14] It is caused by high blood pressure from opiate drugs such as etorphine-hydrochloride (M99®, Norvartis SA, PO Box 92, Isando 1600, Tel: 27-11-929 2387). The problem can be prevented by using azaparone (Stressnil®, Bayer Animal Health SA, PO Box 143, Isando 1600, Tel: 27-11-921 5911) in the dart "cocktail" to decrease blood pressure.

**Agalactia.** Agalactia may occur with capture stress, but the incidence is low (<1%). In one case, agalactia occurred during transport; the cow was injected with 200 IU of an oxytocin derivative (Fentocin®, Virbac) and the elephant was successfully relocated.[5]

**Poaching.** Poaching for ivory accounts for the greatest number of elephant deaths and is not just a recent problem. From 1879–1883 Europe imported 848,000 kg of ivory per year.[15] Elephant numbers in Africa declined from 1,192,300 in 1981 to 622,700 in 1989.[1] Population estimates as of this writing are from 402,000–660,000.[2] Elephants that are wounded during poaching attempts often become quite dangerous. Elephants may also be injured by snares intended for antelope or other wildlife that are hunted for meat.

**Natural causes.** Elephants have no natural animal enemies. Droughts are responsible for the greatest percentage of deaths from environmental conditions. During a severe drought in the Tsavo National Park, Kenya in 1970–1971, 5900 elephants died.[3]

## POPULATION CONTROL

Using contraceptive technology for population control in developing countries where there is a shortage of protein is a controversial issue. However, it is a useful tool on small reserves where every elephant has a name and deciding which elephants to cull would be traumatic for the staff. A contraceptive vaccine produced from porcine zona pellucida (PZP) ova has been used successfully on small game reserves in southern Africa.[17] The initial vaccination is followed by a booster at 3 weeks and then annually. The vaccine is not a hormone and no behavioral changes have been associated with its use.

## DISEASES AND DISORDERS OF CAPTIVE ELEPHANTS

### Infectious Diseases

**Salmonellosis.** Salmonella is discussed in detail in Chapter 11. In Africa, salmonella occurs under conditions of overcrowding and stress. Three syndromes are seen: septicemia, acute enteritis, and chronic enteritis. Watery faces may be contaminated with blood, mucus, or fibrinous material. Elephants may show signs of abdominal pain (hunched posture) and may eat soil. During the acute phase they are anorexic. Neutropenia and elevated fibrinogen are seen in the blood. A series of four fecal cultures are taken as the organism is shed intermittently. Pending results, treatment may be initiated with PeniLA® (Virbac) and an antiinflammatory drug such as Finadyne® (Schering-Plough, PO Box 46, Isando 1600, Tel: 27-11-922 3314). Preventive measures include maintaining a routine to minimize stress and controlling rodents that may act as vectors. Outbreaks have been associated with heavy parasite loads, so routine deworming is recommended.

Elephant areas should be maintained with a high level of hygiene. Infected areas may be disinfected with a stabilized glutaraldehyde (RT 14®, Healthtech Laboratories, PO Box 12285, Villa Nora 1686, Tel: 27-11-805 5703) which is in a slow-release carrier and has a residual effect of up to 14 days.

**Abscesses.** Abscesses are frequently caused by *Staphylococcus* and *Corynebacterium*; however, an antibiogram (culture and sensitivity) is advised to determine treatment. Abscesses that cannot break through the skin will spread laterally and must be drained surgically. Exudate drained from abscesses should be burned to prevent environmental contamination.

### Noninfectious Diseases

**Colic.** Signs of colic may include restlessness, rolling, stretching posture, bloating, lack of defecation, biting of

trunk tip, tenesmus, and groaning. Du Toit[5] described two types of colic in elephants. Spasmodic colic is caused by moldy lucerne (alfalfa) hay and may be treated with Buscopan (Bayer Animal Health SA, PO Box 143, Isando 1600, Tel: 27-11-921 5911). Obstruction colic results from the excessive intake of clay and high-fiber food causing hard fecal balls to form that are difficult to pass. Geophagy (eating soil) is normal among wild elephants, and sodium is probably the stimulus for the behavior.[13] The condition may be treated with a muscle relaxant (Valium®, Roche, 340 Kingsland Street, Nutley, New Jersey 07110, Tel: 973-235-5000)and an antiinflammatory (Finadyne®, Schering-Plough). Colic is prevented by feeding good-quality hay and providing supplemental minerals to prevent elephants from eating soil.

**Choking.** Choking occurs when elephants compete for whole fruits or vegetables. Large oranges may be too big to swallow and may cause an obstruction. The elephant will be unable to drink water and may become dehydrated. Muscle relaxants may be given as a conservative treatment, but usually the foreign body must be removed surgically. The incision must be treated as an open wound to prevent esophageal stricture. Cutting fruits and vegetables into smaller pieces is preventative.

**Nutritional disorders.**

Rickets. Lameness and swollen tibiotarsal joints of one or both rear legs are common signs of rickets in young elephants. The tibiotarsal joint may buckle medially. Treatment is to correct the diet so that it contains 9% crude protein and 1 gram of calcium and 0.5 grams of phosphorus per kg of feed intake. Limit the movement of the elephant and provide adequate bedding material. An orthopedic brace may be used to support an affected joint.

Hypocalcemic tetany. Elephants that are kept indoors for a prolonged period of time may develop a deficiency of vitamin D, which is important for the uptake of calcium. A lack of calcium will cause tetany. The clinical signs are stiffness; nervous symptoms such as eye twitching, uncoordinated movement of the trunk, and pharyngeal paralysis. Calcium borogluconate (Merial, SA, PO Box 5924, Halfway House 1685, Tel: 27-11-315 8001) may be given to "downer" animals. The drip should be administered through a leg vein because calcium is a tissue irritant and may cause sloughing if injected extravascularly. Downer animals must be lifted onto their feet as soon as possible. Care must be taken because they are strong after calcium administration and tend to attack people the moment they are helped onto their feet. When standing, elephants may be treated with 10 mg diazepam (Valium®, Roche) intravenously. This will stimulate the intake of water, to which electrolytes may be added.

Anemia. Long-term chronic deficiency of iron may lead to anemia. Clinical signs include weakness and pale mucous membranes. The hematocrit will be <20. Treatment is ferrous sulphate in the diet or exposure to soil.

Zinc. Excess dietary calcium may cause a deficiency of zinc resulting in skin abnormalities. In some areas—e.g., the Etosha National Park in Namibia—the calcium levels in the water are high because of the calcrete rock formations. There is an imbalance in Ca:P and the wild elephants have brittle ivory. The diet for captive elephants may be corrected by feeding natural bark from browse or Boskos®, (Wes Feeds, PO Box 340, Thabazimbi 0380, Tel: 27-11-777 1330).

**Toxicities.** Avocados contain a cardiac toxin and should not be fed to elephants. Moldy food should also be avoided. Fungi like *Diplodia* in maize and *Aspergillus* in ground nuts will result in nervous and liver problems, respectively. The first clinical sign may be colic.

**Foot conditions.** Inadequate management with poor hygiene causes a condition known as *hoof canker*. It is characterized by a chronic, purulent infection of the skin between the nail and toe. This condition may be treated surgically, but it takes time for the tissue to heal properly. The nails may overgrow in captive conditions and regular trimming is important. If neglected, cracking of the nail may cause lameness or infection. Puncture wounds caused by sharp objects may lead to lameness and infection. Debriding and flushing of the wound with povidone-iodine is necessary together with local and systemic antibiotics. See Chapter 20 for more details. Vaccination against tetanus is recommended for captive elephants.

**Control and management of bulls in musth.** Bulls may be treated with a long-acting tranquilizer such as perphenazene-enantate (Trilafon®, Kyron Laboratories, PO Box 27329, Benrose 2011, Tel: 27-11-618 1544). A total dose of 300–500 mg is given intramuscularly, and the bull is separated from human contact for the duration of musth. Flanagan and Flanagan[6] successfully castrated an adult elephant bull in Zimbabwe.

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# Section II—India

Jacob V. Cheeran and K. Chandrasekharan

## INTRODUCTION

More than 50% of all wild elephants and about 20% of all captive Asian elephants are in India. This corresponds to about 22,000 wild and 3600 captive elephants. The elephant has been an integral part of Indian culture and history through the ages and remains so today.

## INFECTIOUS DISEASES

### Tuberculosis (TB)

The prevalence of TB in captive elephants has not been evaluated, but it is likely due to the intermingling of sick and healthy elephants and humans during festivals, and the high prevalence in the human population. There are anecdotal reports of mahouts (elephant handlers) that have contracted TB from elephants. There is no standardized testing for TB in elephants.

### Wounds

Puncture wounds are common in captive elephants and result from spiked hobbles; sharp, metal-tipped sticks used for prodding; and elephant hooks. Chain injuries to legs occur from protracted tethering during musth, which may last 3 weeks to 3 months.

Pressure sores that may form abscesses occur on the shoulder and hip from lying on hard surfaces and are also common. Most of the wound dressings used are conventional because of the large quantity that is required for cleaning and dressing. Old ulcers and sinuses are packed with magnesium-sulphate-glycerine paste for lymphagogue (drawing) action. Old septic wounds are cleaned initially with hydrogen peroxide. Glycerine acriflavine is used to dress wounds that have a very uneven surface. Indolent ulcers are initially treated with triple sulph (mag-sulph, zinc-sulph, and cupri-sulph). Initial healing is fast, but the superficial (skin) healing takes considerable time.

### Other

There are reports of seroprevalence of leptospirosis in elephants. The authors are aware of two clinical cases; one elephant survived but the other died.

## PARASITIC DISEASES

A list of parasites recorded in elephants in India is presented in Table 35.1. Details are discussed in Chapter 12.

## NONINFECTIOUS DISORDERS

### Impaction of Colon

**Predisposing factors.** Impaction of colon is a common disease condition in captive elephants in Kerala, southern India[2,9] and has been previously reported in other areas.[5,6] This disease results mostly from inappropriate feeding practices. The staple food of captive elephants in Kerala is palm leaves (coconut palm, *Cocos nucifera,* and fishtail palm, *Caryota urens*). Palm leaves are rich in fiber and only rarely are elephants given grass. Hence, in many situations an optimum proportion of browse, grass, and concentrates is lacking in their diet.

The ribs of the coconut palm leaves are difficult to chew and digest, and they form large fecal boluses that

**Table 35.1.** Endo- and Ectoparasites Found in Elephants in India

| Name | Location |
|---|---|
| **Round Worms** | |
| *Murshidia indica* | Intestine |
| *Murshidia murshidia* | Intestine |
| *Murshidia falcifera* | Intestine |
| *Quilonea travancra* | Intestine |
| *Quilonea rennie* | Intestine |
| *Decrucla additictia* | Intestine |
| *Amira pileata* | Intestine |
| *Chonlangium epistomum* | Intestine |
| *Equinurbia sipunculiformis* | Intestine |
| *Bathmostomum sangrei* | Intestine |
| *Grammocephalus varedatus* | Liver |
| *Parabronema indicum* | Stomach |
| *Parabronema smithi* | Stomach |
| *Indofilaria pattabhiramani* | Cutaneous nodules |
| *Indofilaria elephantis* | Probably portal vessel |
| **Amphistomes** | |
| *Pseudodiscus collinsi* | Stomach and intestine |
| *Pseudodiscus hawkesi* | Stomach and intestine |
| *Gastridiscus secumdus* | Stomach and intestine |
| *Pfenderius papillatus* | Stomach and intestine |
| **Flukes** | |
| *Fasciola jacksoni* (liver fluke) | Bile duct |
| *Bivitellobiharzia nairi* (blood fluke) | Portal vessels |
| **Tapeworms** | |
| *Anoplocephala manubriata* | Intestine |
| **Protozoa** | |
| *Trypanosoma evansi* | Blood |
| **Ectoparasites** | |
| **Lice** | |
| *Haematomyzus elephantis* | Skin |
| **Ticks** | |
| *Boophilus annulatus* | Skin |
| *Haemophysalis spinigera* | Skin |
| *Rhipicephalus haemophysaloides* | Skin |
| *Ornithodorus savignyl* | Skin |
| **Fly** | |
| *Cobboldia elephantis* (maggots) | Stomach (gastric myiasis) |

may block the gastrointestinal tract. Providing water at inappropriate times (such as after long, tiring work in the sun without giving sufficient time to cool down) may be contributory. The majority of the religious festivals that display caparisoned elephants take place during the hot months of the year. During the festivals, elephants are exposed to sun in daytime, and inexperienced mahouts water the animal immediately after festivals. This trend results in an increase in the number of impaction cases during the festival season.

Another predisposing factor for impaction is defective teeth. In some areas elephants used for timber hauling pull the logs by holding a vegetable fiber rope in their teeth rather than by a harness as in other parts of Asia. This practice causes the teeth to wear down prematurely leading to defective chewing of coarse foods and digestive problems. Also the proportion of concentrates and roughage in the feed affects the normal transit time in elephants.[7]

According to experienced mahouts and elephant owners, elephants that eat hurriedly are more prone to impaction and colic. There is also another reason behind this hurried eating. Elephants are given palm fronds between two working sessions and they are forced to take their ration hurriedly rather than leisurely, which should be the normal feeding practice for elephants.

Signs of impaction are discussed in Chapter 22. In India, signs may progress until the elephant ultimately ceases to eat and drink and appears dull with a drooping head and closed eyes. On palpation of the body, the sternal region is cold to touch. Dung boluses become smaller and fewer in number and dung boluses may not be palpable on rectal examination. In horses, inadequately masticated or digested feed may move in a retrograde direction for repeated digestion and the same may occur in elephants.

**Biochemical changes.** Hematology and serum chemistry evaluation usually reveals normal values for RBC, hemoglobin, erythrocyte sedimentation rate, WBC, and differential. The hematocrit, BUN, bicarbonate, and lactate levels are generally elevated; chloride, glucose, and potassium are decreased. Sodium, creatinine, and aspartate amino transferase show no significant changes. The hypochloremia and hypokalemia are indicative of a mild metabolic alkalosis. Hypoglycemia also occurs and may be due to a negative energy balance because the animal is not taking feed properly. Liver function is found to be unaltered.[8]

In many instances elephants become dehydrated, especially if the impaction is in the anterior colon, because a large quantity of fluid reabsorption takes place from the posterior part of large intestine. Elephants are tested for the degree of dehydration by pulling the skin between the forelegs and checking for pliability and resilience. Impaction may result in bacterial multiplication and the production of endotoxins. Endotoxins may be absorbed into the body during impaction because the integrity of the intestinal wall is disrupted.

**Treatment.** During the initial stages of treatment, electrolyte replacement (with dextrose saline and other electrolyte solutions) is more important than volume replacement. Elephants are commonly tethered in the open, exposed to the sun. To prevent electrolyte imbalance, the animal should be shifted to a shade area to prevent heat exhaustion and loss of both sodium and chloride ions, which may result in respiratory alkalosis due to panting.

To promote peristalsis, prokinetic drugs such as metoclopramide and cisapride are used. Powerful neuromuscular purgatives, (cholinomimetics like carbachol or neostigmine) should be administered with extreme caution because the margin of safety is narrow.[1] Parenteral alimentation with dextrose, B complex vitamins,

and emulsified fat (parenteral preparation) is usually given on alternate days. When the dung boluses reach the rectum, they are removed manually. Impactions may last for several days and may be fatal. A record period of 75 days was noted in a protracted case of impaction in Kerala. A standard treatment procedure for impaction in Kerala is given below. The dose, rate, duration, and all other parameters should be modified according to the discretion and experience of the veterinarian attending the case:

1. Analgin (matamizole), an analgesic, antipyretic, and antispasmodic (Novalgin®, 500 mg/ml, Aventis Pharma Ltd., Mumbai–16): 60–90 ml IM.
2. Pheneramine maleate, an antihistamine (Avil®, 22.75 mg/ml, Hoescht India Ltd, Bombay): 70–100 ml IM.
3. Calcium panthothenate (D-panthenol, 50 mg/ml, Zigma Laboratories, Mumbai): 50–70 ml IV.
4. Metoclopramide, a gastrointestinal motility stimulator and antiemetic, (Perinorm®, 5 mg/ml, IPCA Lab Ltd., Mumbai-67): 50–60 ml IV.
5. Calcium borogluconate (25%, Rhone-Poulenc Chemicals, Mumbai): 450–900 ml IV.
6. Carbachol 5–10 mg IM or neostigmine/prostigmine to stimulate peristalsis (Neostigmine®, 0.5 mg/ml, Nicholas Piramel India Ltd., Mumbai-12): 3–4 mg IM.
7. Combination of electrolytes/dextrose/fat (in emulsified form, parenteral preparation): a total of 15–25 liters IV.
8. Polybion (B-complex vitamins with vitamin $B_{12}$, (Merck India, Mumbai-18): 50 ml IV.
9. Antimicrobials: chloramphencol succinate (Chloramphenicol succinate®, Lyka Hetero Healthcare Ltd., Mumbai-57): 10–20 g IV daily for 7 days; or ampicillin (Aristocillin®, Aristo Pharma Ltd., Mumbai-53): 10–15 g IV daily for 7 days; or metronidazole (Aldezol®, 500 mg, Albert David, Kolkatta-72): 500–700 ml IV daily for 7 days.

## Eye Injuries

Injuries to the eyes resulting in corneal opacity are commonly seen in elephants in many parts of India. This arises out of two reasons: vitamin A deficiency and traumatic injury. In areas where greens are not available in plenty, elephants are fed with straw alone (hay is not common in India), usually resulting in vitamin A deficiency. Traumatic injuries may result from mahouts hitting the elephant with a stick on the cheek. Often the stick hits the eye causing an inflammatory reaction and opacity. Treatment includes washing the eye with an antiseptic solution, vitamin A supplementation (600,000 units, intramuscularly), and subconjunctival administration of 2 cc Placentrex® (Albert David, Kolkatta-72) on alternate days for 3 weeks (Placentrex is an extract of human placenta; its action is as a nonspecific immunostimulant). The subconjunctival injection is given by inserting a fine needle through the outer aspect of the eyelid and feeling the needle between the fingers to insure that the needle does not penetrate through the eyelid (see Figures 35.4 and 35.5).

## Photosensitization and Adverse Drug Reactions

Both phenothiazines and ketamine may cause photosensitization. The Elephant Tranquillization Team of Kerala Agricultural University has been immobilizing

***Figure 35.4.*** *Administration of subconjunctival injection of Placentrex® (photo courtesy of Dr. Sunil Chawal and Dr. Khyne U Mar).*

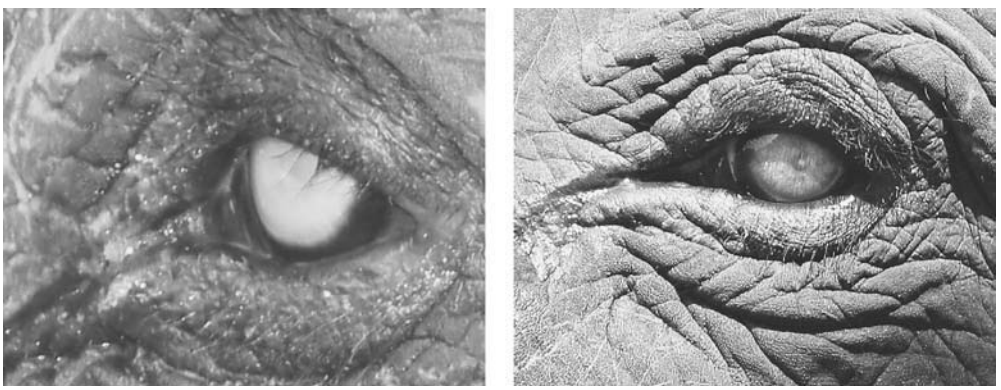

***Figure 35.5.*** *Corneal opacity and conjunctival discharge in a 52-year-old Asian elephant (left) and appearance of cornea 7 days following subconjunctival administration of Placentrex® (photo courtesy of Dr. Sunil Chawal and Dr. Khyne U Mar).*

bulls in musth, which run amuck using xylazine and acepromazine, for the last 25 years. Today, the total number of immobilized elephants is almost 500. Among those animals that were immobilized and exposed to sunlight for a long period after immobilization, some of the elephants developed photosensitization on their back. On one occasion, an elephant that was darted using xylazine-ketamine also developed photosensitization. The area was triangular in shape, starting at the base of the neck. Some elephants that remained aggressive for a considerable period after immobilization had severe local reactions from photosensitization, and in some cases the skin sloughed. Aggressiveness often precluded adequate treatment.

Photosensitization reactions that have been observed in Kerala may be a geographic phenomenon. Kerala is situated at the southern tip of India, close to the tropics, and solar radiation is very high. Flapping the ears, spraying pharyngeal secretions over the body, and mud bath are the usual mechanisms that help the elephant to cool its body. The absence of such activities in a drug-immobilized elephant increases the chances for photosensitization. It is advisable to splash water over the animal after drug immobilization, both to cool off the body as well as to cause arousal. Most immobilizations now use xylazine alone, without adding acepromazine or ketamine.[3]

Another adverse drug reaction that has been noticed is the perivascular effusion of thiopentone sodium when it was given as an anesthetic. In one instance the drug was administered at double the concentration that is recommended for anesthesia. Slight perivascular effusion in the ear vein resulted in phlebitis, cellulitis, and ulcer formation.[4]

Severe gastroenteritis with bleeding in the stomach has occurred after administration of diclofenac sodium, a nonsteroidal antiinflammatory agent used extensively in India. There was one instance in which the animal succumbed. This is often treated with ranitdine, an $H_2$ blocker.

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# Section III—Indochina and Bangladesh

Paolo Martelli

## INTRODUCTION

This section attempts to address veterinary problems in parts of the home range of the Asian elephant about which little is published. Information was collected directly from mahouts (elephant handlers) and veterinarians and is largely anecdotal. It is hoped this information will inspire further interest in elephant husbandry and medicine in Indochina.

Elephants have played a major role in the culture and history of the great civilizations of Asia and have been "domesticated" for thousands of years. In recent times, mechanization has rendered elephants almost obsolete for customary functions such as warfare, transport, and logging. An explosive human population and habitat conversion to agriculture are driving the Asian elephant to extinction.

The traditional trade of the mahout is vanishing. Many captive elephants are unemployed and (un)attended in poorly managed camps with deficient infrastructures. The only sector that seems to bear some prospect is tourism. This may regrettably lead to a time when the Asian elephant may be present only in zoos and tourist facilities.

## INDOCHINA

### Status of free-ranging and captive populations

Surveys of wild elephant populations are infrequent and comparison of data is of limited value because of differences in sampling methods.[2] It is estimated that 57–81 wild elephants live in Vietnam,[6] 200–500 in Cambodia,[4] (personal communication, Chheang Dany, Cambodia, March 2005), and 200–500 in Laos.[10] Herds are small and isolated.[3] Wild elephants are nearly extinct in Vietnam.[1,11] Pressure on wild elephants is on a scale unparalleled elsewhere.

Between 1964–1973 (Vietnam War) the U.S. obliterated large extents of forest in the elephant-rich areas of Cambodia, Laos, and Vietnam. In the last 3 decades, rapid population and economic growth has resulted in extensive land reclamation for agriculture and industry, further reducing elephant habitat. Poaching is common. Elephants are shot for food, bones, nails, and hair. Between May 2001–December 2002, 32 elephants were killed in the southern Cardamom mountains in Cambodia.[13] Human-elephant conflict is escalating, legal and illegal logging are intense, and land encroachment omnipresent.

Captive populations have also declined. Estimates are 864 in Laos,[5] 150 in Cambodia (personal communication, Chheang Dany, Cambodia, March 2005), and 40–165 in Vietnam.[3] Threats to captive elephants are extinction of mahoutship, unemployment, shortage of food, lack of veterinary care, lack of breeding, and aging populations.

### Veterinary problems

Data on diseases in the wild is lacking. Most captive elephants in Indochina do not have access to modern veterinary medicine. Mahouts, using traditional or intuitive treatments, usually administer veterinary care. Elephant veterinary expertise in Vietnam lies with the veterinarians of the Hanoi and Saigon zoos. In Cambodia and Laos there are few veterinarians and no veterinary schools. The Phnom Tamao Zoo and Rescue Center in Cambodia and the Ban Kheun Zoo in Laos employ full-time veterinarians and house 4 and 3 elephants, respectively. The tourist operation, Compagnie des Elephant d'Angkor in Cambodia holds 18 elephants, employs a professional elephant manager, and has weekly veterinary visits. An elephant clinic is being established in Laos in a district where 57 logging elephants reside.

#### Infectious diseases.

Viral and bacterial diseases. Little is known about infectious diseases of elephants in Indochina. Foot and mouth disease type O is endemic but has not been reported in elephants. There were no reports of poxvirus or papillomatous lesions. DNA from tissues of a 30-month-old calf that died suddenly in Cambodia was extracted at the Institut Pasteur du Cambodge and analyzed at the Institute for Zoo and Wildlife Research (Berlin). It revealed a novel strain of endotheliotropic herpes virus (personal communication, Thomas Hildebrandt, San Diego, August 2004). Tuberculosis is present in humans, pet and laboratory macaques, and livestock, but there were no reports in elephants. Hemorrhagic septicemia and anthrax occur in cattle and buffaloes and must be included in the differential diagnosis of sudden death in Indochina.

**Internal parasites.** *Murshidia falcifera, Bunostonum, Ancylostoma, Trichostrongylus, Strogyloides, Parabronema Paramphistomum,* and other trematodes were reported from feces and necropsies in Vietnam. The malarial blood parasite *Plasmodium vivax* was identified at the Institut Pasteur de Saigon in an elephant calf suffering from fatal fluctuating edema, and an unidentified microfilaria was seen in a cow that also died of the disorder. Anthelmintics used prophylactically in captive elephants include mebendazole (25 g/elephant), fenbendazole (25g/elephant), benzimidazole (10 mg/kg), or pyrantel (25 g/elephant every 3 months) or ivermectin (0.2 mg/kg PO or SQ every 6 months). Village elephants are rarely treated.

#### Noninfectious diseases.

Poor-doers. This ambiguous syndrome was frequently reported. Signs include decreased stamina, depression, slowed response to commands, poor appetite, and skin and nail problems.

Mahouts often send the elephant off to the forest when signs occur, in the hope that they will recuperate spontaneously. Treatment may include 10–30 ml IM of multivitamins (Stressvitamins®, Vetoquinol SA 70204 Lure cedex, France); 100 ml of vitamin B12 IM (Catosal®, Shinil Chemical, Seoul Korea) and vitamin C (5–20 g IM). A vine locally known in Laos as "Keu Kao Hong" (*Tinospora crispa*) is fed to increase stamina.

Diarrhea. Diarrhea is uncommon among logging elephants that feed in the forest, but it occurs in zoo, circus, and tourist elephants fed excesses of sugar cane and bananas. Treatment includes oral charcoal tablets (10–20), atropine (0.02–0.06 mg/kg IM), liniment rubs, 7 billion units of *Lactobacillus sporogenes*, 5% dextrose IV, and rehydrating salts. Antibiotics that may be used include oxytetracycline (20 mg/kg IM), sulfaguanidine (20–30 mg/kg IM sid), chloramphenicol (6.25g/calf PO or 20 mg/kg IM sid), trimethoprim-sulfonamide (60–70 g/adult elephant or 30 mg/kg PO), nifuroxazide (Ercefuryl®, Synthelabo France), ciprofloxacine or enrofloxacine (2mg/kg IM sid), tetracycline (10 mg/kg IM sid), ampicillin (10 mg/kg IM sid), and metronidazole (4 g/calf IM sid).

Traditional treatments from Vietnam include table salt, boiled dipterocarp, ashes of straw or grass, and

ground horns and skin of serow (*Naemorhedus capricornis*).[3] In Laos, traditional treatments include coconut leaves, roots of water ferns, and tamarind. Prayers are written on thick cloths that are ceremoniously burnt to ashes, added to the mixture above, and fed to the sick elephant (personal communication, Sukwan and BounHom, Laos, April 2005).

Constipation. Constipation is treated by manual removal of feces or with laxatives such as sodium polystyrene sulfate (Kionex, Paddock laboratories Inc., Minneapolis MN, USA). A traditional preparation of ground roasted soapberries (known as Bô Kêt in Vietnam) in water and instilled per rectum combined with liniments of ground garlic in rice alcohol rubbed on the abdomen 2–3 times a day was reported effective (personal communication, Thuy VT, Vietnam, October 2004).

Colic. Treatment of colic includes exercise and abdominal massages using camphor or garlic liniment rubs, atropine (0.02 mg/kg IM), dexamethasone (200–400 mg/elephant), diclofenac (750 mg/subadult), biolactine, sulfaguanidine (20–30 mg/kg IM sid), or metronidazole (3–5 mg/kg IM sid for 2–6 days).

Anorexia. Anorexia is considered a sign of grave illness. Treatment may include abdominal rubs, vitamin B complex, and vitamin C IM. Antibiotics and antiinflammatory drugs may also be given.

Geophagy. Elephants are often observed to ingest wet soil. Diarrhea and expulsion of parasites often follows and in Vietnam this is viewed as a self-medicating behavior.

Malnutrition. Signs of malnutrition are regularly encountered in Cambodia where calves are wild-caught and raised on soymilk, rice water, boiled rice, bamboo shoots, or bananas. The few that survive often have limb or spine deformities.

Successful treatment in two cases consisted of correction of the diet, ampicillin (4 g/day for one month IM), a nonsteroidal antiinflammatory (NSAID) drug such as ibuprofen (3200 mg PO sid every for 20 days), vitamin D (12 cc/day IM), calcium phosphate powder (12 g/day PO), calcium carbonate (7.5 g/day PO), vitamin C (5g/day), and vitamin E (3500 IU/day). Vitamins and calcium supplements were given for 3 months.

Foot care and problems. Routine foot care is not practiced in Indochina. Cracks, growth rings, and flaking of the nails may reflect nutritional or metabolic problems. Snares are common in Cambodia and may cause serious injuries to the legs and trunk. Scars from chains or ropes are often visible. Because of the lack of access to veterinary care, logging elephants sometimes lose several months of work or remain disabled from complications of untreated infections. Wounds from land mines are occasionally seen.

Lameness. Lameness may be due to excessive exercise, walking on paved roads, joint disease, sepsis, or trauma. Treatments include antibiotics, steroids (dexamethasone 200 mg/elephant IM sid) or NSAIDs (phenylbutazone, 1–3 mg/kg IV; acetaminophen, 12,500 mg/elephant PO q 24 hours for 3–6 days). Vitamin C (0.5 to 5 g/elephant/day), vitamin B complex (10–30 cc/elephant), calcium powder (1–3 g/day/elephant), and massage with liniments may also be used. A liniment prepared by mixing ground fried *Ficus* aerial roots in rice wine was described in Vietnam.

Trauma, wounds, and abscesses. Wounds on the chest, abdomen, or back are typically caused by straps or chairs (howdahs). Wounds on the head and ears are caused by the ankus or ear ropes (ropes, sometimes combined with a metal hook that are placed in or around the ear to control the elephant). Tail wounds may be inflicted by other elephants.

Wounds are cleaned with various solutions depending on availability and personal preference. Topical applications of tetracycline spray or penicillin powder are common. The following antibiotics were used: amoxicillin (Aescamox® LA, Aesculaap, Boxtel NL) (1.5 g/calf or 20 mg/kg IM q 24 hours), penicillin (120 ml/adult IM q 24 hours), ampicillin (15 mg/kg q 24 hours), trimethoprim-sulfadiazine (30 mg/kg q 24 hours PO), and oxytetracycline (20 mg/kg IM). Termite nests in ammonia and various boiled medicinal plants are used as traditional treatments in Vietnam.[3] Abscesses are allowed to rupture and are then drained and treated like open wounds. An antiinflammatory proteolytic enzyme extracted from silkworms (Serratiopeptidase, Arthropower® Insect, Biotech Co, Daejeon, Korea) (20 tabs every 24 hours for a month) is reported useful to reduce swelling (personal communication, Thy N, Cambodia, October 2004). Herbal compresses are commonly applied.

Some elephants in Cambodia appear dark, almost black, from the application of an herbal solution of crushed leaves from a tree locally called "snoul" (*Dalbergia nigrescens*) (personal communication, Gavin Bourchier, Cambodia, March 2005) that is used as a sunblock. In Southern Laos elephants appear black from the volcanic mud they wallow in that also acts as a sunblock and fly repellent.

Eye problems. Eye injuries may result from branches or other vegetation during feeding. Saline wash and application of antibiotic eye drops or ointment are the usual treatment. Traditional remedies include powdered seashell blown onto the eye and solutions of herbs in water and rice alcohol sprayed in the eye. *Pasteurella multocida* sensitive to penicillin, ceftazidime, gentamicin, quino-

lones, and rifampin was isolated from an elephant with conjunctivitis in Cambodia.

Dental problems. Most dental problems occur in older elephants and receive little attention. Long tusks are not desirable for logging. Ivory is cut so that it protrudes from the trunk by a hand-span or less. Removed fragments are sold. Exposed pulp is treated as an open wound.

Collapse. The terminal sign of old age in elephants usually is collapse followed by death within 4–24 hours. An adult bull with elevated serum lipids and total and esterified cholesterol collapsed and was suspected to have an ischemic cardiac infarction. He was treated with a substance (clofibrat) that lowers steric lipids, triglycerides, and cholesterol. The elephant recovered fully in one week (personal communication, Xuan, Vietnam, October 2004).

Scaly dermatitis. A chronic dermatitis of three years duration and unknown etiology has been documented in three elephants in Cambodia. The lesions are illustrated in Figure 35.6. Microscopic examination of fresh scales with Giemsa, KOH, Diff-Quick® (Dade Behring, Dudingen, Germany), and lactophenol failed to reveal fungal hyphae, spores, or parasites, but a gram stain revealed organisms resembling *Dermatophilus congolenis*, a bacteria that causes streptotrichosis in cattle and horses. The condition has not spread to other elephants in the herd. The condition is essentially a nonpruriginous hyperkeratosis-orthokeratosis (accumulation of keratinized layers without nuclear retention) (Martelli unpublished). Topical antiseptic and antifungal agents have been unrewarding although improvement was noted when cajuput oil (*Melaleuca cajeputi*) was applied (personal communication, Gavin Bourchier, Cambodia, March 2005).

**Reproduction.** Expertise in breeding is limited. Traditionally breeding is not desired in Vietnam and Cambodia. In Laos a birth is a rare event and cause for great joy. Lao mahouts believe that elephants require much privacy to breed. The demand for calves by zoos and tourist operations is increasing. Temporal secretions are considered a sign of estrus. Priapism was observed in Vietnam.[12] Bulls in musth are isolated or chained with free access to water. Food is reduced drastically to weaken the animal and limit the duration of musth.

**Sedation.** The use of sedation is rare. Xylazine is the drug of choice. Doses of 50–500 mg were reported for procedures including translocation, treatment, and euthanasia. Etorphine and other opioids are not available though their use is not illegal. There was one documented anesthesia with Thiopenthal (3.3% at 20 mg/kg IM) for surgery to remove a mass. Time to lateral recumbency was 30 minutes. Caffeine (1,250 mg) was administered IM to assist recovery. The elephant was standing 4 hours after induction (personal communication, Dr Thuy, Vietnam, October 2004). Euthanasia is illegal in Laos and legal in Vietnam and Cambodia.

## BANGLADESH

### Status of Free-Ranging Elephants

Only 196–227 elephants are believed to live in this densely populated country. Of these, 83–100 migrate regularly from Burma and India.[8] Most herds are composed of 5–8 elephants with a male-to-female ratio of

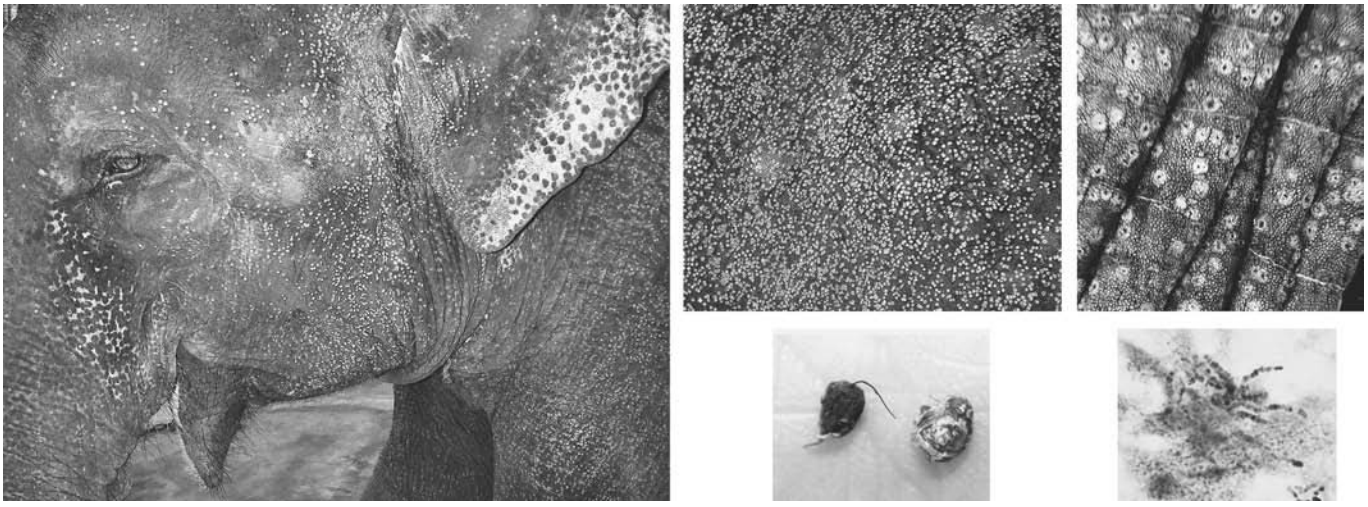

***Figure 35.6.*** *Generalized scaly dermatitis resembling streptotrichosis (dermatophilosis) in an elephant in Cambodia. The top-center and top-right photos are close-ups of the lesions. The lower-center image is a crusty plug that was removed manually. The lower-right photo is a gram stain showing organisms that have the characteristic appearance of* Dermatophilus congolensis. *A definitive diagnosis was not established (photo courtesy of Hank Hammatt; photomicrograph by Friends Childrens Hospital in Siem Reap, Cambodia).*

1:3.[8] The main threats to wild elephants are human settlements and lack of tree cover and food.[8]

Between 1997–2002, human-elephant conflict caused >$500,000 (US) in damage. In that same period, 162 settlers and 22 elephants were killed. Tribal people are unaffected, as they know the elephant routes and avoid placing crops or homes nearby (personal communication, Kanh, Bangladesh, December 2004). Local residents are generally sympathetic toward elephants and favor solutions such as elephant watches to protect crops. Despite this, crop-raiding elephants may be injured or killed by firearms, poison-tipped spears and arrows, homemade bombs, firearms, battery acid, petrol bombs, and poisoning. Poaching is uncommon and snares are rarely employed. Bangladesh is a signatory of MIKE (Monitoring of Illegal Killing of Elephants). Deaths due to accident or disease are occasionally documented.

### Status of Captive Elephants

There are approximately 90 captive elephants in Bangladesh.[7,8] Many of these were captive born. The last Khedah (elephant roundup, also known as *khedda or keddah*) was in 1967. There are about 25 privately owned elephants (a sign of wealth and prestige that is a declining phenomena) and 14 elephants in circuses (a declining trade), logging (a vanishing industry), zoos (three cows) or in the tourism sector (an emerging industry). The Bangladesh Forest Industry Development Corporation (BFIDC), a subsidiary of the government Forest Department, was dissolved in 2004 when logging ceased for lack of suitable forests. Of the 13 elephants owned by BFIDC, 6 were returned to the forest department to be retrained for tourist activities.

Mahouts are knowledgeable about nutrition, husbandry, reproduction, and traditional medicine. The training techniques observed by the author were remarkably gentle, especially in comparison to those practiced in other parts of Asia. Training of captive-born elephants begins at an early age. Typically one mahout takes charge of one elephant, but experienced mahouts handle several. Captive elephants are taught to stand still, raise the trunk, open the mouth, lie in sternal and lateral recumbency, bow, move in all directions, and present all feet to allow work on the soles and nails (see Figure 35.7). Traditional mahoutship is in crisis. Logging has ceased and better-paid, less dangerous jobs are available. One can anticipate that within the next decades this unique, time-honored knowledge will have disappeared, without leaving a substantial record.

## VETERINARY PROBLEMS

### Infectious Diseases

Infectious diseases of wild and captive elephants include anthrax, foot and mouth disease (FMD) type O, hemorrhagic septicemia, and tetanus. Elephants under veterinary supervision are vaccinated yearly against tetanus, anthrax, and hemorrhagic septicemia, but not against FMD.

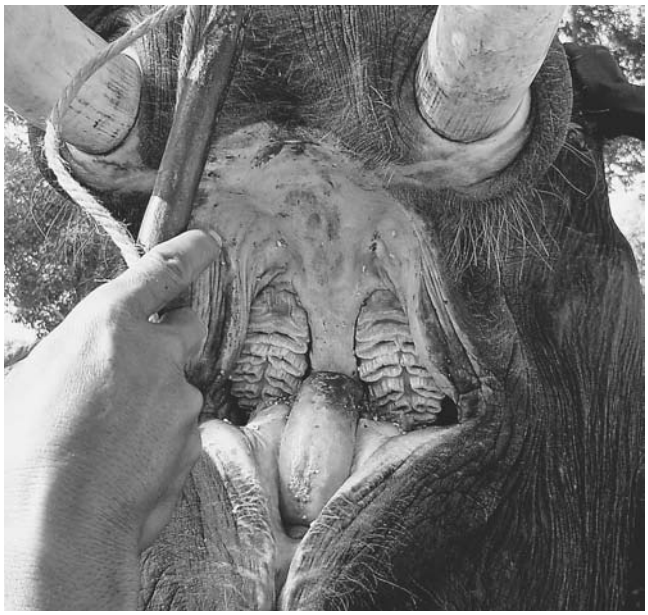

***Figure 35.7.*** *Elephants in Bangladesh are trained to open their mouths for routine examination (photo by Paolo Martelli).*

**Foot and mouth disease.** Multiple interdigital vesicles on all four feet were seen in a cow with FMD at the Dhaka Zoo in 2002. The cow refused to allow topical treatment because of intense pain. Fluid was aspirated from the vesicles, and FMD type O was isolated. No oral lesions were noted and foot lesions healed in 45 days (personal communication, Dr. M.S. Iqbal and Dr. N.C. Banik, Bangladesh, December 2004).

**Anthrax.** In 1981 an adult bull at the Dhaka Zoo died acutely of anthrax[9] (personal communication, Dr. N.C. Banik and Dr. M.S. Iqbal, Bangladesh, December 2004). A laboratory technician developed cutaneous anthrax from handling the samples (personal communication, Dr. N.C. Banik and Dr. Hira, Bangladesh, December 2004). Anthrax has also been diagnosed in free-ranging elephants.

**Abscesses.** A paste of turmeric, salt, and chili or a solution of sodium hydroxide and salt is applied to developing abscesses. Abscesses are lanced with a sharp knife sterilized over a flame or in boiling water and then flushed with boiled water, chlorhexidine gluconate (Savlon®, Novartis), and boric acid. The cavity is packed with cut mimosa leaves, cooked rice, and coal to speed coagulation, increase exudation, and stimulate granulation. The abscess is kept open until healing is complete.

**Wounds.** Wounds are common among logging elephants. Mahouts use boiled salt water or chlorhexidine

to wash the wound several times daily. Myiasis is common. The powdery content of cell batteries (alkaline manganese) is sprinkled on the wound as a fly repellent and to kill emerging maggots. Veterinarians are consulted only for major injuries.

**Fungal dermatitis.** Fungal lesions may be difficult to differentiate from scars. A shiny appearance to the discolored lesion, a response to topical antifungal agents, and rapid repigmentation are characteristic of fungal lesions, whereas scars do not regain pigmentation. The most widely used antifungal agent is engine oil.

## Parasitic diseases

Intestinal nematodes are common. Anthelmintics used include albendazole (2 g/adult PO), mebendazole (10 mg/kg or 2g elephant PO), ivermectin injectable (0.2 mg/kg SQ), and a combination of levamisole and triclabendazole (Endex®, Novartis Keppel towers, Singapore) (100ml/ton PO). Elephants from the forestry department are wormed 3–4 times a year. The elephants at the Dhaka Zoo are wormed twice a year. There were no definitive identifications of the parasites. Mahouts note that the ingestion of mud precedes parasite-related watery diarrhea and present the elephant for treatment when they observe this sign.

**Lice.** The traditional treatment for lice consists of soaking dried tobacco leaves in water and applying to affected areas (nicotine is a known insecticide).

## Noninfectious Disorders

**Diarrhea.** In Bangladesh bananas are considered a common cause of diarrhea and are seldom fed. Elephants in circuses are subject to frequent diet changes and poor food quality. These animals are often diarrheic, malnourished, and parasitized (personal communication, Dr Saifuddin, Bangladesh, December 2004). Treatment of diarrhea includes 2–4 liters of normal saline administered rapidly IV 2–3 times a day and pediatric oral rehydrating salts in water or delivered in balls of rice, banana leaves and molasses. Sulfatrimexazole (5–10 mg/kg) PO for 5 days or longer may also be used. Mahouts offer rice with grilled garlic to speed recovery and reduce malodorous flatulence.

**Constipation.** Excessive ingestion of fibrous plants (e.g., banana stems) is believed to predispose to constipation. Treatment includes walking and ordering the elephant to lie down and stand up repeatedly to help expel fecal boluses. Vegetable oils are given orally and feces are manually removed from the rectum.

**Colic.** Flatulent colic often follows diarrhea. Grilled garlic in rice is offered. Charcoal is not used in the traditional treatment of flatulence; however, mahouts reported that elephants consume ashes from the fires (personal Communication, Mhd Hussein, Mhd Farouk, Bangladesh, December 2004).

**Ventral edema and ascites.** Calves raised on goat or cow milk may develop ascites and ventral edema, which may have a nutritional or parasitic etiology. Furosemide (1mg/kg IM) has been used unsuccessfully as an attempted treatment for ventral edema in adults. Ventral edema resolves spontaneously.

**Glossitis.** Glossitis of unknown etiology has been observed in certain elephants and may be recurrent. Signs may last for 1–2 weeks and include ptyalism, dysphagia, and a swollen, painful tongue. An allegedly successful treatment used by mahouts is the application of a paste of tumeric, salt, and mustard oil.

**Muscular pain.** Muscle pain is common among logging elephants. Hot water compresses, camphor liniments, and massage are applied and elephants are rested or exercise is reduced. A traditional antiinflammatory preparation contains an extract of acacia locally known as "khor," two types of resin locally called "Musabbe" and "Smirish," tobacco leaves, black pepper, garlic and pods from a plant locally called "musaranga." These ingredients are ground and boiled together and applied topically (see Figure 35.8).

**Foot care.** Foot care is the responsibility of the mahout. A sharp knife is used to trim overgrown nails or cuticle. To trim the sole, melted road tar is applied, allowed to dry, and then scraped off together with the overgrown sole. Sole punctures by thorns and splinters are common. Elephants may extract these foreign bodies themselves, or mahouts will do so with sharp bamboo sticks and pliers.

**Tusk injuries.** Cracks in the tusk are widened with a file to prevent extension. Exposed pulp is packed with mustard oil and generally treated as an open wound.

**Eye problems.** A mixture of mustard oil and salt is painted around the eyes as a fly repellent and reportedly to prevent bacterial growth (see Figure 35.8).

**Reproduction.** Elephant managers in Bangladesh have more experience with breeding than in many other Asian countries. Captive breeding has been carried out at the BFIDC and Forestry department for over a century.[7] Behavioral observations are used to detect estrus and may include a temporal discharge lasting 4–5 days, reduced obedience, short temper, restlessness, and seeking out other elephants, especially males. Mating, when not witnessed, is confirmed by locating traces of dried sperm around the vulva. The urine of a gravid elephant is reported to cause a fresh patch of grass to die within 24 hours after a single urination. The urine becomes

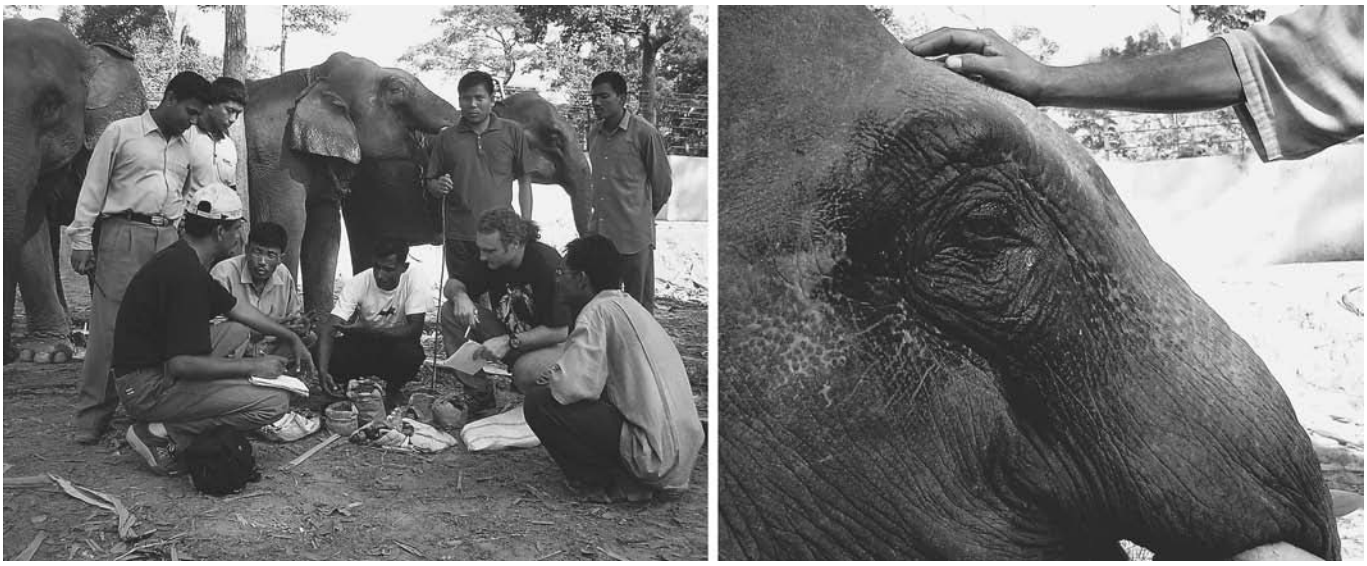

***Figure 35.8.*** *Left: Mahouts at the Duhazala Safari park demonstrating traditional medications used in Bangladeshi elephantology to the author (author kneeling, second from the right). Right: Salted mustard oil is applied around the eye as a fly repellent (photo by Paolo Martelli).*

greenish, darker, and more turbid in gravid females. Mammary development with a coconut-milk–like secretion begins 4–6 months before birth. Closer to parturition the female will rest her head against trees and shows signs of abdominal discomfort, such as lying down and trumpeting. There were no reports of aggressive maternal behavior toward the newborn calf.

**Musth.** Logging bulls that are worked hard do not generally come into full musth. Bulls in musth are chained. Food is not reduced, but bulls may eat less.

**Euthanasia.** Euthanasia is illegal in Bangladesh. Local veterinarians are intent on educating the authorities to the humane aspects of euthanasia.

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# Section IV—Indonesia

Yudha Fahrimal and Retno Sudarwati

## INTRODUCTION

The Sumatran elephant (*Elephas maximus sumatranus*) is the most endangered of the Asian elephant subspecies. Estimates of only 2800–4500 wild elephants in 47 fragmented populations were reported in a population analysis in 1993.[25] In 2002 the estimate of wild Sumatran elephants had declined to 2440–3354.[24] Of 12 elephant populations identified in the 1990s in Lampung Province in southern Sumatra, only three still exist. Recently, detailed surveys indicate that although two of these herds have sustainable numbers (498 and 180, respectively) the third is not likely viable.[7] The total population of Indonesian captive Sumatran elephants in 2004 was 482 animals. A burgeoning human population, conversion of elephant forest to agricultural use, and poaching have led to increased human-elephant conflicts on Sumatra.

### Elephant Training Centers

The Government of Indonesia elected to capture elephants as an attempted solution to human-elephant conflict. Between 1986 and 1996, approximately 520 elephants were captured and placed in Elephant Training Centers (ETCs) to be trained for logging, agriculture, and tourism.[10] For the most part these uses of elephants have not materialized except for a small number used to patrol national parks.

Approximatley 300–400 captive elephants reside in six major ETCs. Veterinary problems and care have been the subject of numerous reports.[8,9,10,11,12,15,26] Common problems shared by the ETCs include insufficient food for elephants and lack of veterinary supplies and training. Adequate funding is an ongoing problem and most centers are at or over capacity. Clinical problems include marginal nutrition, intestinal parasitism, and infection from wounds.

## INFECTIOUS DISEASES

There are no reports of infectious diseases in wild Sumatran elephants. Tetanus may occur in captivity due to poor husbandry. The first sign noted is difficulty moving the extremities. If seizures occur, the prognosis is guarded. Antitetanus serum injection is very important for the wounded elephant with potential for *Clostridium tetani* infection.

*Pseudomonas aeruginosa* is a major causative agent of severe ulcerative keratitis. Corneal damage may result from direct injury or wind damage sustained during transport on open flatbed trailers (see Figure 35.9). The ulcers may be characterized by rapid liquefaction of the corneal stroma, which may progress to perforation.[6] Corneal opacities may be bilateral and may progress to perforations and blindness. The cornea will stain positive with fluorescent dye. Treatment includes antibacterial agents, collagenase inhibitors, and corneal protectants. Loose exudate is debrided from the cornea, and dilute iodine is applied with a sterile cotton-tipped swab. A subconjunctival injection of gentamicin (5 mg) is given. Tobramycin is used if there is resistance to gentamicin. Systemic antibiotic injections may also be given. A 3rd eyelid flap has also been used successfully (see Figure 35.10).

Mortality in newly captured elephants may result from septicemia secondary to infected dart wounds.[16,17] Improved hygiene during darting, wound cleaning, the administration of antibiotics, and the inclusion of a veterinarian on the capture teams have been suggested.

## PARASITIC DISEASES

The tropical climate of Indonesia is conducive to parasitism. *Fasciola sp.* is the major trematode, and infection may be accompanied by elevated liver enzymes (SGPT


***Figure 35.9.*** *Elephants are commonly transported in open trucks and may sustain corneal damage from wind. Positioning elephants to face the back of the truck may help prevent such injuries.*

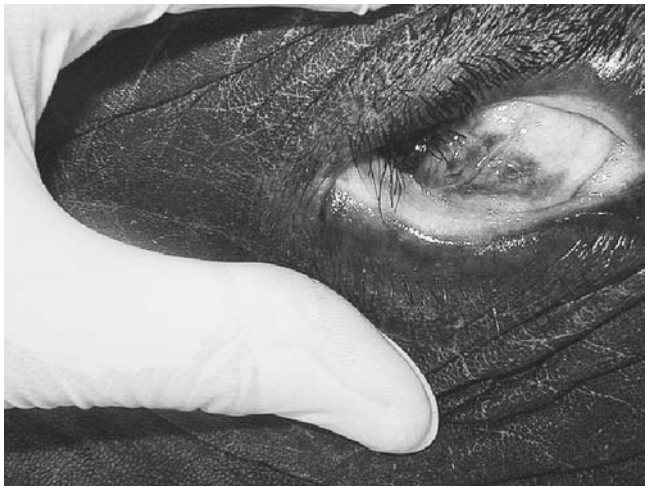

***Figure 35.10.*** *A nictitating membrane (third eyelid) flap was applied to an elephant with ulcerative keratitis. The nictitating membrane acts as a bandage to protect the cornea and facilitate healing.*

and SGOT). Albendazole (5–10 mg/kg) or triclabendazole (Fascinex®), 10 mg/kg are used for treatment. Treatment may be repeated monthly in severe cases.

Strongyles and strongyloides are the usual nematodes found in Sumatran elephants. Treatment is with albendazole or oxfendazole. Levamisole is used with caution because severe tremors have been observed following oral administration at a dosage of 8 mg/kg.

*Murshidia falcifera*, two species of trematodes (*Hawkesius hawkesi* and *Pfenderius papillatus*), and *Cobboldia elephantis* were found in three Sumatran elephants that died of clostridiosis.[14]

*Cobboldia elephantis* (botfly larvae) may be found in the stomach and may occur in large numbers. *Cobboldia* parasites were once found in the abdomen of a Thai elephant that died and was necropsied in Indonesia.

## NONINFECTIOUS DISORDERS

### Trauma, Wounds, and Abscesses

Newly captured elephants may sustain leg wounds from chains and neck wounds from the *kah,* a yoke made of wood and wire that is placed around the neck to restrict movement. Wounds may also occur during training. The training process is harsh and elephants often lose condition during the process.[9] Abscesses resulting from dart wounds sustained during capture may take weeks to resolve and if left untreated may progress (over a period of weeks or months) to a necrotizing fasciitis that is often fatal (see Chapter 18, Figure 18.7).

Mortality that has occurred during translocation has been attributed to tranquilizer overdose,[1] but sternal recumbency during transport on flatbed trailers may also be a factor.

Because of damage to crops and humans caused by elephants, elephants may also become victims—usually poisoned or shot by angry villagers.[2,3,4,27] Injuries to limbs or trunks may result from snares (see Figure 35.11). Poaching of elephants for ivory also occurs.[5]

### Nutrition

Nutrition is an ongoing concern for elephants in the ETCs. Captive elephants are tethered in the forest to graze during the day and those tended by caring mahouts are moved several times to new areas. At most ETCs, elephants are brought back to the base camp at night. Supplemental feed (banana stem and coconut palm leaf primarily) may be given at this time. In the author's experience (Fahrimal) 26 elephants at one ETC that were monitored over a 2-year period either lost weight or grew at less than 40% of the rate documented for other Asian elephants. Half of the elephants at this camp were anemic (PCV <32) the first year tested, and 40% were anemic the following year. Poor nutrition is the likely underlying cause, although intestinal and/or blood parasites may be contributory.

### Other

**Edema.** Edema may be seen in the submandibular or ventral abdominal areas. Poor food quality may be a

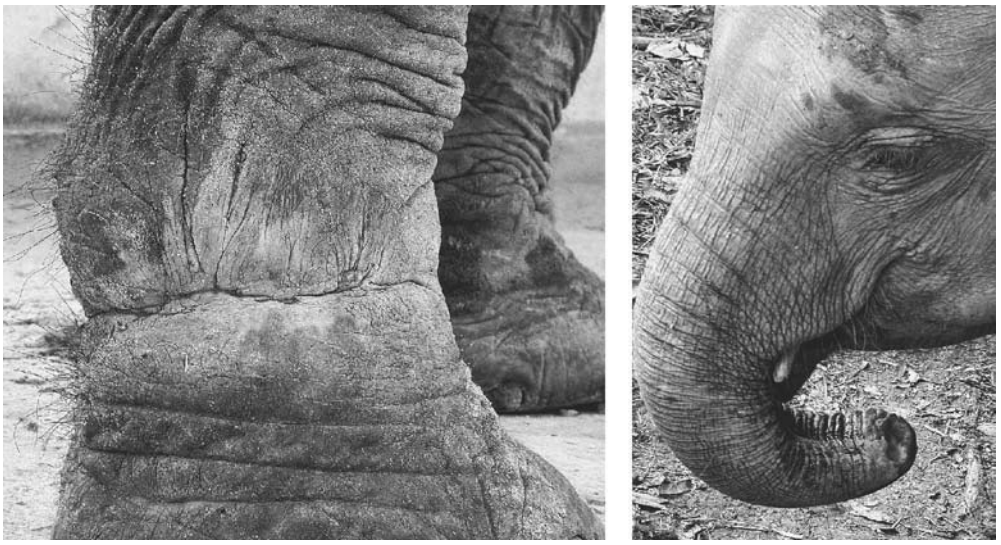

***Figure 35.11.*** *Crop raiding elephants may fall victim to snares placed by villagers. Young elephants are at particular risk and may sustain injuries to trunks and lower limbs. Damage to the distal end of the trunk (right) may be fatal for a young elephant. Snare injuries to legs may heal with early intervention (left).*

cause and hypoproteinemia may be associated. Ventral edema is also seen in cows that have recently given birth. Increasing the protein in the diet may help (see Chapter 18, Figure 18.10).

**Bloat and constipation.** Bloat and constipation occur primarily due to unsuitable food with too high a fiber content, and cases occur most often during the dry season. Soapy warm water enemas are given as treatment.[21] Bloat is also treated with antiflatulen (Tympasol®, Bremer Pharma GMBH, Germany) orally. For an adult elephant 300 ml of Tympasol® mixed with 3 liters of warm water is given. Vitamin B complex may be given as supportive treatment.

**Seizures.** The author (Sudarwati) has experience with one case of an elephant with recurrent seizures. At 6 months of age the elephant sustained a deep wound to the occipital area from a tiger bite. The elephant would lean his head against the wall repeatedly until it was 3 years old. Seizures began when the animal was 5 years old, usually lasting for 10 minutes and occurring every 2 months. Cold weather and stress (e.g., environmental factors, competition for food, and human interaction) would trigger seizures. During seizures, the animal would become stiff in the left extremities, display aggressiveness, and become laterally recumbent. No serum chemistry abnormalities were recorded. At 10 years of age blindness was apparent in the left eye. Treatment and prevention have included muscle relaxants (diazepam 0.01mg/kg), vitamin injections, a warm environment, good-quality food, and minimizing stress.

**Hand rearing.** It is important for orphaned calves to receive colostrum within the first 24 hours. If colostrum is not available serum may be given (see Chapter 16). An elephant calf was successfully hand-reared at Taman Safari Indonesia using the following formula: brown rice, powdered milk (Lactogen 2®, Nestle, New Zealand), and sugar with 3:3:1 ratio. The brown rice is cooked, filtered, and mixed together with the other ingredients and water. The volume of the formula is increased as the calf grows.

The formula is given every 2–3 hours until 2 months of age. After that, boiled rice is given without filtration, and banana juice is introduced. At 3 months of age, apples and carrots are introduced. Grass is introduced when the calf is 1 month old.[13]

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# Section V—Malaysia

Vellayan Subramanian

## INTRODUCTION

### Status of Wild Elephants

In the 19th century, prior to the arrival of the colonial powers, the introduction of firearms, and the large-scale conversion of forests to other land use, elephants (*Elephas maximus hirsutus*) occurred throughout peninsular Malaysia. Today, elephants in the wild are found in small, scattered groups in nine states. The Department of Wildlife and National Parks (DWNP) manages elephants in the wild in peninsular Malaysia. In East Malaysia elephants are not found in Sarawak, but in Sabah the sighting and capture of rare pygmy elephants has provided impetus for studies to establish its species status. The future of wild elephants outside protected areas is precarious given the country's rapid pace of development and desire to increase its human population substantially.[5] There are approximately 1200 wild elephants in Malaysia.[10]

### Status of Captive Elephants

Captivity of elephants in Malaysia began in the 1960s. Zoo Negara Malaysia obtained two adult male elephants from the Great Indian Circus in 1964. Subsequently, Burmese and Thai elephants were brought in to work in capturing wild elephants by the Department of Wildlife and National Parks. Between 1960 and 1970, elephants were kept by royalties, a practice that has been abandoned. Private ownership of elephants is now illegal. There are 44 captive elephants in Malaysia, housed mostly in zoos.

**Kuala Gandah Elephant Sanctuary.** The DWNP established the Kuala Gandah Elephant Conservation Center in 1989. Kuala Gandah supports research and conservation and promotes public awareness of the elephant's plight in Malaysia. The center also serves as the base for the Elephant Relocation Team.

## TRANSLOCATION

In 1974, the DWNP established the Elephant Management Unit to initiate a translocation program. The Elephant Relocation Team is dedicated to locating, subduing, and translocating wild elephants from areas where

their habitats are encroached by plantations to suitable habitats, such as Taman Negara National Park[2]. Since 1974 the team has relocated more than 500 elephants.[7] The program was developed with assistance from four male khoonkie elephants (accompanied by their mahouts) that were brought from Assam, India.[9]

The Unit has developed a successful technique for the translocation of wild elephants that combines Mela-Shikar (capture using ropes), as practiced in Assam, and modern chemical immobilization techniques.[6] Xylazine and etorphine hydrochloride combined with acepromazine maleate (Immobilon®, Vericore Ltd., U.K.) are the primary agents used for capture. Translocation involves chemical and mechanical aspects, including tying up, removal to the loading site, loading onto the lorry (truck), unloading, and release. Postcapture identification and postrelease monitoring are important. Translocation procedures used in Malaysia, Sri Lanka, and India have been reviewed.[8]

## INFECTIOUS DISEASES

### Bacterial Diseases

*Salmonella blockley* caused a fatal infection in four zoo elephants in Malaysia.[1] The author has isolated pathogenic *Escherichia coli* that were unable to be serotyped in Malaysia from young calves. *E. coli* may cause colibacillosis in young elephants, particularly in those that have not received sufficient colostrum. Young who suffer from malnutrition are prone to the disease. Colibacillosis may be treated with antibacterial drugs such as Kaolin Mixture with Belladonna (Bangalore, India, 500 gm for 2,000 kg) and Suspension Lepromide (Lomide®, Siam Bheasach, Co., Thailand, 5 ml/10kg, body weight bid or tid). Alpha-heamolytic *Streptococcus* and *Staphylococcus* spp. have been isolated from abscesses. *Proteus* spp., *Bacillus subtilis, Klebsiella* spp., and *Staphylococcus pyogenes* were isolated from an orbital tumor.

### Viral and Other Infectious Diseases

Viral diseases are rare among elephants in Malaysia. There is only one recorded case of elephant poxlike lesions at Zoo Negara. It was treated with tumeric powder (Anitha's Mfg. and Trading, Malaysia) mixed with Acriflavine (Acriflavine HCl, BP, India) and Eusol®. However, we were unable to confirm the viral isolation because we lacked the facilities.

Fungal infections of the nails and cuticles are common in elephants housed in moist, damp areas. They are treated topically with 1% potassium permanganate and subsequent dipping in 5% formaldehyde for 30 minutes for a period of 1 week.

### Wounds and Abscesses

Superficial skin wounds in captive elephants may result from social fights, hooks, self-inflicted enclosure features, accidental falls, and foreign bodies.[11] Topical preparations commonly used include Margosa Oil® (AK Samy Enterprise, Malaysia), turmeric powder, Himax® ointment, Gusanex® (dichlorofenthion Wokwel PVT. LTD., Singapore), and Zoosamex® spray. Urine burns (resulting from improperly slanted floors) are treated with potassium permanganate and acriflavine.[11]

Abscesses may be treated with daily application of tincture of iodine, 1.8% (Bexton Vet Pharma, Australia). An alternate treatment is to drain the abscess and pack it with gauze soaked in Eusol. Injectable, long-acting antibiotics such as Ampicillin (Bicolistine®, Pharmaniaga Manufacturing, Malaysia, 1–3 gm daily in divided doses, IM or IV), Gentamicin (Gentamed, Medochemie LTD, Limssol, (Cyprus, Europe), 2ml/kg) or chloramphenicol (KRKA, Novo Mesto, Yugoslavia) 20–30 mg per animal may be used.

## PARASITIC DISEASES

### Ectoparasites

Infection with external parasites is common in the wild and captivity. The elephant louse (*Haematomyzus elephantis*) was recorded in peninsular Malaysia by Jeffery.[3] Ticks (*Amblyomma* sp.) are typically found on the perineum, upper thigh, shoulder, and behind the ears of wild-caught elephants. They can be removed manually.

The blood-sucking horsefly (*Tabanus* sp.) is also common. The bites from these flies are painful and irritating to the elephant and result in the formation of nodules on the surface of the skin. Elephants in the wild mud wallow to rid themselves of the flies. Screwworm flies (*Chrysomya bezziana*) lay eggs around open wounds and the maggots burrow and feed on tissues. Maggots must first be removed from the wounds, which are then washed with chlorinated lime, boric acid (Eusol Solution, Safire Pharma., (M) Sdn. Bhd.). Pure chloroform is used to kill maggots that penetrate deep into the muscles (Zoosamex spray®, Trichloro HD 2% W/W, Wokwel Ptd. Ltd., Singapore). Presently the drug of choice for ectoparasite treatment is 1% ivermectin (Yuanzheng Pharma, China, 0.02 ml/kg). A repellent may also be applied.

In captive zoo elephants, the vulva area is prone to myiasis from screwworms. Douching with 0.5% potassium permanganate and external application of Himax® ointment (Natural Remedies, India) or Zoosamex® is used for treatment.

Periodic cleaning of vaginal secretions is preventive.

### Endoparasites

Stress predisposes to endoparasitism. Elephants have died in captivity from liver flukes and ascariasis. Ventral edema may be an indicator of endoparasitism. The endoparasites encountered most frequently in Malaysian zoo elephants are *Strongylus* spp. (88%), *Eimeria* spp. (9%), and *Strombidium* spp.(3%).[4] Intestinal ciliates commonly isolated were *Triplumaria hamertonii* and *T. selenica*.[4]

Other commonly identified nematodes include *Strongyloides* spp., *Murshidia falcifera,* and *Murshidia mushida*. Trematodes that have been isolated from the intestinal tracts are *Pfenderius papillatus* and *Paramphistomum* spp.

### Blood Parasites

Microfilaria of *Loxodontofilaria asiatica* has been detected in the blood of a captive female elephant.[13] Filariasis caused by *Indofilaria patta* has been reported to cause hemorrhagic dermatitis.[5]

## NONINFECTIOUS DISORDERS

### Postcapture Trauma

After capture and while waiting to be transported to the release area, the captives are kept free of the influence of drugs. It is during this time that self-inflicted injuries may occur, primarily to the legs and neck. Chains may cause lacerations with subsequent abscessation. Subcutaneous abscesses associated with puncture wounds from darts used during immobilization have also been observed.

During transportation over long distances, elephants may partially recover from the effects of the xylazine sedative (Sedazine®, Fort Dodge Animal Health, Fort Dodge, USA). They have been observed to use their trunks to knock down the side wall of the truck or to break it with their tusks. Such actions may result in tusk damage.

A thorough understanding of the causes of injuries and the subsequent resulting problems is necessary in order to prevent or reduce them during the process of translocation. All animals should be thoroughly examined soon after capture and promptly treated for lacerations or abrasions prior to translocation. After immobilization a high dose of broad-spectrum antibiotics should be administered.[5]

### Trauma Associated with Leg Chains

Chains are a source of trauma for both captive and wild elephants. It was a common practice to chain captive elephants at night. This caused wounds, abscesses, and arthritis.[12] It is now recommended in Malaysia not to chain elephants during day or night. The use of electric fencing to prevent escape is highly encouraged. This practice is followed at the Singapore Zoo. The author recommends the adoption of this practice for all elephants in captivity.

### Arthritis and Foot Disorders

Overworn soles may result from excessively moist conditions. Elephants may be lame, reluctant to move, and have a tender sole upon palpation. It is corrected by keeping the feet dry. Pododermatitis is treated with potassium permanganate and 2–5% formalin followed by warm water. Topical antibiotics may also be used. Split nails are trimmed and a topical hoof dressing applied (Corona®, Summit Industries, Inc., GA, USA). Overgrown cuticles are treated with daily applications of vegetable or mineral oil. This will soften the cuticle, and the elephant will rub off the excess.[12]

### Gastrointestinal (GI) Disorders

GI disorders are common in elephants fed large amounts of ripe fruit. Infectious etiologies include *Salmonella* spp., *Escherichia coli*, *Pseudomonas* spp., and *Clostridium* spp. These can be successfully identified, and subsequently treated, by culture of the feces.

Constipation or impaction are common and results from feeding high-fiber foods such as long banana stems that are not easily digested and may become impacted in the colon or cecum. The diagnosis may be confirmed by rectal examination. Treatment includes the administration of cathartics, soapy enemas, analgesics and/or antipyretics, diet change, and antibiotics.

Colic, bloat, and abdominal discomfort may be caused by the diet, toxins, or foreign bodies. This may be treated with Timpol® preparation (Natural Remedies, Bangalore, India), 300–500 gm per adult elephant, weighing about 2 tons, mixed with Gingely Oil (or any vegetable oil) followed with cathartics, analgesics, and/or antipyretics or diet changes. An alternative method is mixing one packet of Timpol® (100 gm) with 3 kg of cooked molasses rice and 5 bottles (100 ml each) of dimethicone (Blotsil®, Nevketan Pharmacology, India) for each adult elephant; this mixture is administered orally. Exercise is essential.

### Toxicities

Arsenic compounds are used to kill weeds in plantations. Elephants are attracted by the salt content in the chemical compounds and will readily eat the poison. The animal usually dies a few miles from the plantation. There was one case of elephant mortality from arsenical compound that was used by workers of the forest department to control forest undergrowth. Four elephants were found dead after taking the poison.

Deliberate use of lineate was discovered in one oil palm plantation. Three elephants that ate it died immediately.

### Miscellaneous Disorders

Dental problems in captive Malaysian elephants are rare but may occur if tusks break or are trimmed too short. Cutting the tusk back too short can expose the pulp, resulting in bacterial infections and pus formation. The treatment given is daily dressing with 35% hydrogen peroxide solvent and cauterizing with silver nitrate crystals.

Excessive, continuous lacrimation has been observed in some zoo elephants fed pineapple and pineapple waste. Foreign bodies such as sand and dust may also cause excessive lacrimation. The first captive-born calf in Zoo Negara suffered damage to the right cornea and was treated symptomatically by washing the eye and ap-

plying Nicol® Eye Ointment (UPHA Pharmaceutical Manufacturing (M) Sdn. Bhd, Malaysia).

The zoos that keep male elephants in captivity control musth in a very conventional method. When the male comes into musth, he is chained and food intake reduced for the entire period of the musth, eliminating all sugar and protein from the diet but maintaining fiber. In aggressive bulls, xylazine is administered.

## ACKNOWLEDGMENTS

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# Section VI—Myanmar

Khyne U Mar

## INTRODUCTION

The elephant is of great cultural and historical significance in Myanmar and of major economic importance to the timber industry. Over 4000 elephants work in logging under the government-operated Myanma Timber Enterprise (MTE) or private ownership.[2,3] Estimates of wild populations vary from 3000 to 10,000[13] but these are declining due to habitat fragmentation, developmental activities, unsustainable agriculture practices, and poaching.[14]

## ELEPHANTS IN CAPTIVITY

There are two major elephant keeping systems in Asia, which may be defined as *extensive* and *intensive*.[7,8] Working elephants in forest camps of Myanmar, Assam, and south India live in extensive keeping systems, and elephants kept by temples or private owners live under intensive keeping systems. Under intensive keeping systems, elephants are kept more-or-less individually, fed prepared fodder, and tethered at night.

Extensive keeping is the traditional management system in Asia that dates back many centuries. Under this system the daily species-specific activities are high. Elephants are hobbled and released into the forest at night to forage and interact with tame and wild conspecifics. Stereotypies are absent or rare.

Most elephants are wild caught; a smaller number are captive born.[4,6,10] Subadults (4–5 years) are preferentially captured because they are more easily tamed.

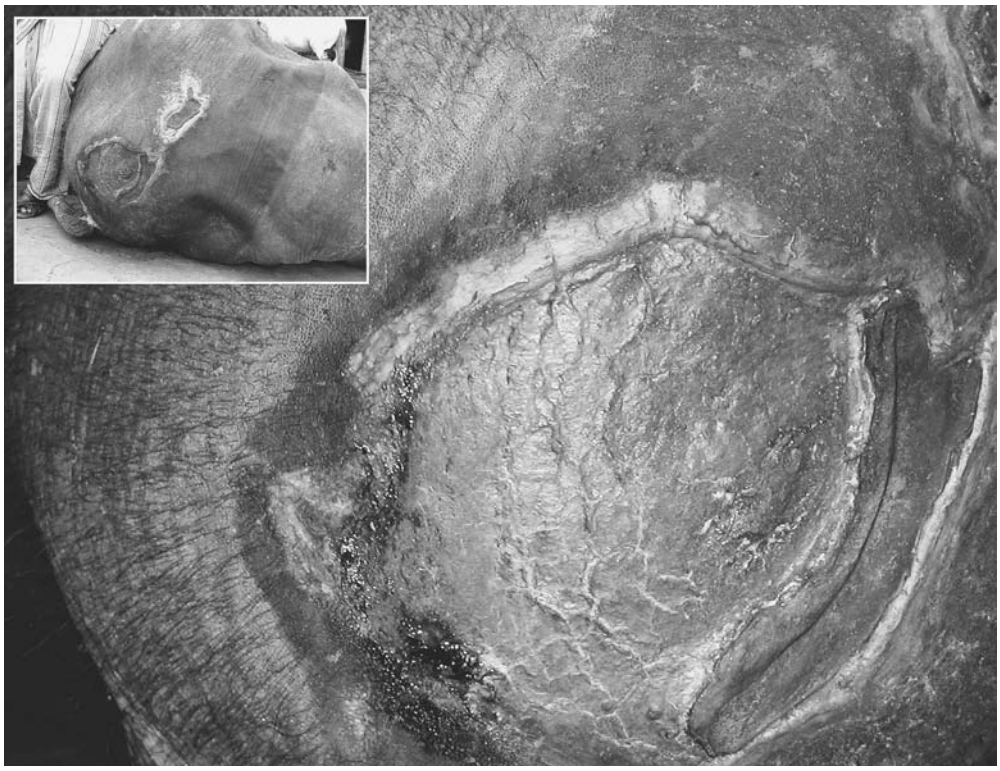

***Figure 35.12.*** *Sunburn on the head of an Asian elephant. A typical erythematous peeling of the skin occurs first, followed by exposure of unpigmented subdermal tissue with a necrotizing central area.*

Taming is done during the cool season between November and January.[6] Each elephant is assigned a permanent registration number, a mahout, and a log book, in which its biodata (sex, temperament, musth, mating, calving, veterinary intervention, etc.) are recorded. All government-owned elephants are chemically branded using a corrosive paste of kaolin, caustic soda and glycerin.[4,5] Branding distinguishes tame from wild or privately owned elephants. The latter have their own branding marks.

In each calendar year, elephants work 7 months, 5 days/week, 5–8 hours/day. They are allowed 12–16 hours/day of free-range foraging at night in the working months of June to January and longer hours during the hot nonworking months. Elephants are kept in mixed herds of adult males and females and calves that are functionally analogous to the social structure of wild herds. Pregnant females and mothers with suckling calves do not work until calves are a year old.

Working elephants are usually handled by two mahouts. Bulls in musth and aggressive elephants are assigned an extra man armed with a spear.[1] Elephants >18 years are placed in the work force until the retirement age of 55. After retirement, they spend their time roaming and foraging under the care of one mahout.

Timber elephants are cared for by 25 veterinarians and 34 veterinary assistants appointed by MTE. Veterinarians oversee dragging gear; selection of camp sites; and the accommodation, transportation, general welfare, and training of mahouts. There is no intervention with wild elephants, but information is collected to monitor risk assessment for camp elephants.

## Noninfectious Diseases

### Trauma.

Accidents. Most camps are located in difficult terrain. Accidents that may occur include choking; drowning; being caught in forest fires and quicksand; falls from cliffs; strangulation by its own chain; snakebite; and attacks by older siblings, wild elephants, or tigers.

Sunburn and heatstroke. Prolonged exposure to direct sun may result in sunburn (see Figure 35.12). Soothing ointments are applied topically. Heat stroke, caused by overexertion combined with high ambient temperature and humidity, may occur with no warning signs. Treatment includes copious cold water enemas containing 1% table salt; applying cold water to the head, neck, spine, axilla and belly, pouring water into the mouth; and providing shade. Affected animals frequently die. Intravenous fluids and steroids (prednisolone sodium succinate, 1 mg/3kg, or dexamethasone, 1–2mg/5kg) may be given.

Sprains and fractures. Working elephants may sustain sprains, fractures, and dislocations by stepping on soft ground that collapses under their weight, slipping while descending steep hillsides, or getting a foot caught between rocks or logs. Falling logs may also cause injury. Young elephants that sustain simple fractures are placed

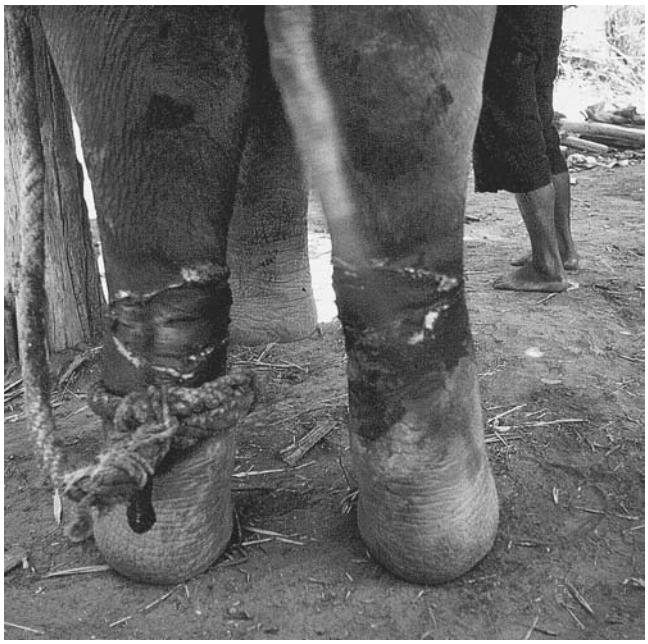

***Figure 35.13.*** *Rope burns sustained during training.*

***Figure 35.14.*** *Dragging gear used with logging elephants in Myanmar. Lard is applied to the breast band to prevent abrasions.*

in a sling and hand-fed. Hot herbal compresses are applied to the affected area. Dislocations have poor prognosis. If there is no indication of recovery in 3 months, euthanasia is the only choice.

**Wounds and abscesses.** Recently captured elephants may develop rope burns on their legs and neck during the breaking procedure (see Figure 35.13). Shallow skin wounds and abrasions usually heal rapidly in healthy elephants with little or no attention. Breast bands, dragging gear, and saddles may cause abrasions.

Well-designed gear, customized to the measurements of the individual elephant, prevents such injuries. All gear is inspected by the veterinarians and is kept scrupulously clean. Lard is applied to breast bands before they are placed (see Figure 35.14). Neglected skin wounds with continuous irritation from ill-fitting gear may ulcerate. Treatment consists of debridement and topical antibiotic ointments. Hot herbal compresses are applied to hasten maturation of abscesses. Abscesses are lanced, drained, and flushed with an antibiotic solution.

**Constipation and impaction.** Constipation or impaction may be precipitated by inadequate access to water, sudden diet changes, excessive mud eating, grains (e.g., sticky rice), or highly fibrous fodder (e.g., straw, twigs, barks, uncut banana stems).

Old elephants (>60 yr) with worn-out molars may not masticate properly. Treatment includes manual removal and warm soapy enemas. Cholinergic stimulating agents, such as carbachol (carbachol amine, 5–10 ml/elephant, IM) may induce peristalsis. Carbachol may cause bradycardia, pinpoint pupils, sweating, excessive lacrimation, bronchial secretions, dyspnea, coughing, and vomiting. Short acting antihistamines (e.g., pheniramine or chlorpheniramine, 500–2000 mg IV or IM, depending on age) will counter these effects. Nonsteroidal antiinflammatory drugs (NSAIDs) in equine dose and large volumes of balanced IV fluids (~50–70 L/day for an adult >4000 kg elephant) and 10% calcium borogluconate (400–800ml) may be given. Feed is restricted until the impaction is relieved.

**Foot problems.** Working elephants have ample opportunity to wear nails and soles naturally and foot problems are rare. Cracked soles and heels may occur in wet conditions (e.g., working in the river or during monsoon). Signs include lameness, pain, erosion, and ulceration. Treatment includes debridement, power flushing with an antiseptic solution, topical antibiotics, dry flooring, and rest. Elephants that drag heavy logs may have vertical splits in hindlimb nails from gripping the ground. Foreign bodies (stones, wood chips, thorns) that become embedded in the sole may cause pain and lameness. Treatment includes removal, footbaths of dilute (1:1000) potassium permanganate and topical copper or zinc sulphate sprays.

## Infectious Diseases

### Parasitic.

Intestinal parasites. Recorded parasites include the nematodes *(Murshidia, Quilonia, Amira, Decrusia, Equinurbia, Chiniangium, Parabronema,* and parasites under the order *Strongyloidea*). Strongyles are the most common parasite. Elephants stressed from recent capture, training, overwork, or malnourishment may shed large num-


***Figure 35.15.*** *Gastric myiasis (bots).*

bers; healthy elephants are usually asymptomatic. Loss of appetite, dullness, depression, and ventral edema are seen with heavy loads. Calves with heavy infections may have swollen abdomens. Oral albendazole (7.5 mg/kg) or fenbendazole (3.75–5.0 mg/kg) is given quarterly.

Gastric myiaisis (bots) is common during the rainy season. Signs include loose stool, mouth breathing, and mud eating in mild cases and colic, diarrhea, anorexia, and anemia in severe cases. Mahouts are instructed to clean the areas around the elephant's mouth and tushes/tusks daily to remove fly eggs. Camphor oil or neem oil (*Azadirachta indica*), may be smeared around the base of the tusks as a fly repellent. Subcutaneous injection of 1% ivermectin (1 ml/50 kg) or 5–20 mg trichlorfon (Neguvon, Bayer) orally is used for treatment (see Figure 35.15).

Paramphistomiasis occurs especially in the rainy season. Signs include anorexia, severe diarrhea, and high mortality in young elephants. Older (>17 yr) animals may harbor numerous adult worms but develop resistance to reinfection. The large, clear, operculated eggs are readily recognized, but in acute infections there may be no eggs in the feces. Albendazole (7.5 mg/kg) for 3 consequent days is effective. Young animals are most affected by *Fasciola jacksoni* and may die in severe cases. Albendazole (7.5–10 mg/kg), triclabendazole (7.5 mg/kg), and oxyclozanide (7.5 mg/kg) are used for treatment.

Cutaneous filariasis. Filariasis causes a chronic progressive dermatitis and is prevalent among timber elephants. Mortality is negligible, but there is significant economic loss of working capacity.[11] Clinical signs include restlessness, exercise intolerance, dry skin, boils, and submandibular and ventral edema. Motile and dead microfilaria may be retrieved by squeezing the boils found on the navel, lower thighs, toes, and heels—places elephants may not reach for scratching and areas most often neglected by mahouts during bathing. Diagnosis is by microscopic observation (10× objective) of motile parasites in unstained thick smears of nocturnal blood samples, collected between 21:00 hrs and 03:00 hrs. Quantification is essential to assess morbidity and monitor the efficacy of treatment. A simple field method developed by Myanmar veterinarians is to place a drop of blood on a clean slide and then use a needle or syringe tip or the corner of another slide to draw the blood out into 3–4 thin, parallel horizontal lines. This restricts parasite movement during counting, which must be completed before the smear dries. If no filaroids are found, the smears are air-dried, stained with Giemsa, and reexamined. Maximum counts reported/smear are 232[11] and 370.[12] The presence of <5, 5–20, and >20 parasites/smear are regarded as light, medium, and heavy infestations, respectively. Ivermectin is effective (0.1 mg/kg SQ) and may be given every 4–6 months for heavy infestations.

Hemorrhagic dermatitis. Tissue-invading larvae of biting warble flies (*Oestradae)* penetrate the skin immediately after hatching and cause irritation as they migrate. Small boils form, and blood-tinged or purulent fluid discharges when the mature instar emerges. The distribution of lesions is similar to cutaneous filariasis. Treatment with topical, parenteral, or orally administered ivermectin or lotion containing 1% trichlorfon (Neguvon, Bayer) is effective.

Lice. Heavy infestations of elephant lice (*Haematomyzus* spp.) may cause dermatitis, pruritis, and dry scaly skin. In an attempt to relieve irritation, elephants often rub their eyes, sometimes by using a stick, and may cause corneal rupture and blindness (see Figure 35.16). Treat-

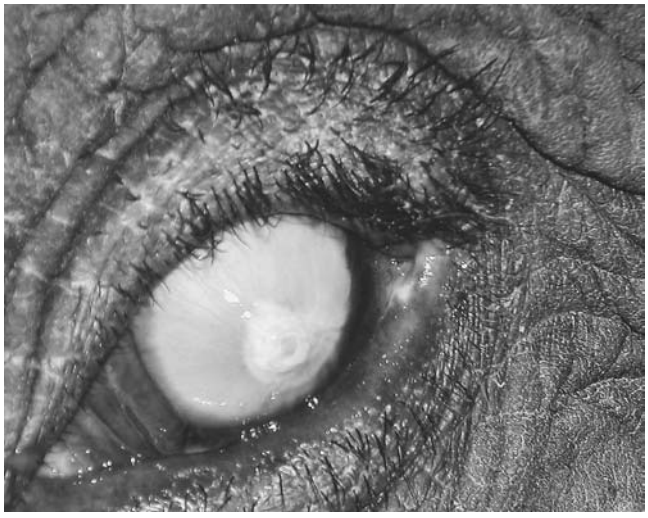

***Figure 35.16.*** *Corneal rupture resulting from scratching secondary to lice infestation around the eyes.*

ment with topical or oral ivermectin is effective. Daily bathing and brisk scrubbing using a pumice stone, coconut skin, or scrub made of the herbal creeper called *su-yit (Acacia pennata)* (found abundantly in the tropical forest) is preventative.

## Bacterial Diseases

**Anthrax.** Sporadic outbreaks of anthrax may occur during April, May, and early June. Although mortality is high in domestic livestock, elephants rarely succumb. Elephants are vaccinated annually with 1 cc of an attenuated vaccine used for horses and mules (Sternes).

**Enterotoxemia.** Enterotoxemia caused by *Salmonella* and *Clostridia* spp. may occur in young elephants. Antibiotic choice is based on stool culture and sensitivity results. Antibacterial and fluid therapy should be prompt and vigorous.

**Tetanus.** Tetanus is reported occasionally. Treatment includes draining and cleaning wounds, administering high doses of penicillin or other broad-spectrum antibiotics, and hand-feeding easily digestible feeds (see Chapter 11 for details).

**Tuberculosis (TB).** Confirmed deaths from TB have occurred in working elephants, and the prevalence is probably higher than reported due to poor surveillance systems for both elephants and humans. Trunk exudates are cultured and sensitivity testing performed (see the tuberculosis section in Chapter 11 for details).

**Hemorrhagic septicemia (HS).** The clinical signs of HS are similar to anthrax, and acute cases may be fatal. A provisional diagnosis is based on clinical signs, gross lesions, and disease prevalence in the area. Diagnosis is confirmed by identification of the organism, biochemical and serological tests, nonserological tests (e.g., the acriflavin flocculation test) or PCR.

Treatment with a wide range of antibiotics is effective. Vaccination is a major control measure, and all elephants >6 months (except pregnant females) receive a 5 ml SQ inoculation of alum-precipitated hemorrhagic septicemia vaccine (APHS) vaccine twice a year.

## Viral Diseases

**Foot and mouth (FMD).** The epizootiology of FMD in Myanmar is complicated by lax control activities among the country's livestock population of 11.7 million cattle, 2.6 million buffalo, 4.8 million pigs, and 2.2 million sheep and goats.[9] The last FMD outbreak occurred in 1999. Elephants may be exposed when cattle or buffalo invade their foraging areas. Clinical signs are discussed in Chapter 11. In some elephants, the entire sole may separate. Treatment in Myanmar consists of high doses of broad spectrum antibiotics to control secondary bacterial infection and nonsteroid antiinflammatory agents. Nursing care consists of providing clean, dry, dust-free flooring; clean drinking water ad libitum; and easily digestible feed (e.g., rice porridge, bananas) that is hand-fed. Cold potassium permanaganate solution (1:5000) is used for footbaths and to clean the tongue and trunk-tip lesions.

**Endotheliotrophic herpesvirus.** Herpesvirus may have caused sudden death in young elephants (<10 years). Antemortem signs included lethargy, anorexia, edematous swellings of the head and thoracic limbs, oral ulceration, and cyanosis of the tongue.

**Other.** Rabies and elephant pox are rarely reported.

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# Section VII—Nepal

Sunder Shrestha and Kamal P. Gairhe

## INTRODUCTION

Nepal has both resident and migratory wild elephants. Wild elephants are distributed from the eastern to the western border. A rapidly growing human population and its need for land development have led to an increase in human-elephant conflict. As a result, elephants are mostly confined to protected areas. However, their movement extends beyond protected areas.

### Free-Ranging Population

Until 1960 there were large numbers of elephants throughout the entire lowland forest area of Nepal. As a result of a massive human resettlement program the forest cover was extensively cleared and the elephant population dwindled to about 100–138 individuals (personal communication, Narendra Babu Pradhan, Wildlife Officer, Department of National Parks and Wildlife Conservation, DNPWC, His Majesty's Government of Nepal, April 2005). Based on their movement, Nepal's free-ranging elephants comprise four subpopulations.[3]

**Eastern population.** The eastern population consists of temporary migrants from the neighboring state of West Bengal. The total subpopulation varies between 10–15 individuals and is mostly seen during the rice-harvesting season (September to October).

**Central population.** The central population is composed of 25–30 resident elephants within the Parsa Wildlife Reserve. Some splinter groups of this population have found their way into the adjoining Royal Chitwan National Park, as well as into the buffer forest along the park.

**Western population.** The western population currently is between 45 and 50 individuals. With new recruitment, it has become the largest subpopulation in Nepal.

**Far-western population.** The far-western population is the least stable and includes temporary migrants from neighboring India. The current herd is composed of 12–18 elephants.

### Captive Population

As of this writing, Nepal has recorded 174 captive elephants.[3] A total of 86 elephants reside in hattisars (elephant camps). The Department of National Parks and Wildlife Conservation (DNPWC) of the government of Nepal maintains six hattisars, and another is managed by the King Mahendra Trust for Nature Conservation (see Figure 35.17). The remaining 88 captive elephants are kept by the private sector, mostly by hotel operators within and around the protected areas. Elephants are used mainly for ecotourism.

***Figure 35.17.*** *Housing for elephants at the Royal Chitwan National Park (courtesy of Dr. Gretchen Kaufman).*

## INFECTIOUS DISEASES

### Viral Diseases

**Foot and mouth disease (FMD).** Naturally occurring FMD has been recorded in Nepalese elephants. Signs include vesicles and erosions in the mouth and on the feet, lethargy, inappetance, salivation, and high fever (39°C; 103°F). A longitudinal separation of the skin on the ventral part of the trunk was also noted. Type O virus was isolated from one of the outbreaks.[4] No mortality has been reported in elephants. Treatment includes broad-spectrum antibiotics and washing of lesions with an antiseptic solution such as potassium permanganate. Elephants usually recover after 2–3 weeks.

**Rabies.** Based on a history of exposure to a rabid dog and clinical signs, suspected cases of rabies have been reported but not confirmed. Signs included lethargy, loss of appetite, cessation of water intake, unsteadiness, aggressiveness, and restlessness. One elephant also died after showing the signs. Secretion from temporal glands was observed in another elephant. Some elephants fell and were unable to stand again. Elephants at risk for contact with rabid animals are vaccinated with Nobivac-R® or Raxa Rab® (Indian Immunologicals, Rakshapuram, Kothaguda), 2 ml SQ. These vaccines are intended for domestic animals and their efficacy in elephants is unknown.

### Bacterial Diseases

**Salmonellosis.** Salmonellosis is one of the common bacterial diseases observed. Signs include anorexia and heavy foamy diarrhea, some times with bloody mucus. Urination may decrease in elephants that refuse to drink. No deaths due to salmonellosis have been reported. Treatment includes pheneramine maleate (Avil®, Hoechst India Ltd., Bombay, India), 0.2 mg/kg IM; trimethoprim and sulfadiazine (Biotrim Vet®, Ranbaxy Laboratories Ltd., New Delhi, India), 20–30 mg/kg IM; metamizole (Analgin®, Wockhardt Pvt. Ltd., Bombay, India), 40–45 mg/kg IM; and electrolyte solution (Rintose®, Wockhardt Pvt. Ltd., Bombay, India), 1500 ml IV or per rectum. Elephants respond well when treatment is instituted early.

**Tuberculosis (TB).** Two elephants with signs of progressive wasting, intermittent fever, and anorexia had characteristic TB lesions postmortem. Histopathology confirmed the diagnosis. Tuberculosis is under further investigation.

**Tetanus.** Anecdotal reports of tetanus have been described in elephant camps; however, confirmed cases have not been reported. Tetanus toxoid (Adsorbed Tetanus Toxoid BP, Serum Institute of India), 4 cc IM, is given to pregnant females after calving in harsh conditions or traumatic parturition, elephants involved in vigorous forest work with a potential for deep puncture wounds, and elephants with wounds from chain tethering. The effectiveness of vaccination is unknown and needs further study.

**Wounds and abscess.** Swollen, painful, hot abscesses are formed under the skin due to infection from pus-forming bacteria such as *Streptococcus, Staphylococcus, Corynebacterium, and Pseudomonas* spp. Abscesses are seen after contusions, chafing wounds, parasitic invasion, and general debility. Abscesses are drained and irrigated daily with an antiseptic solution. Large abscesses are also treated with parenteral broad-spectrum antibiotics.

**Pododermatitis (foot rot).** Foot rot is common in some camps. The sole of the foot becomes worn and susceptible to bacterial infection. Treatment consists of improving hygiene, washing the soles of the feet daily with a 5% copper sulfate solution, a 10% formalin solution, and painting the affected area with Stockholm Tar. Severe cases are also given broad-spectrum parenteral antibiotics. Overgrown soles, cracked soles, cracked heels, overgrown nails, and split nails are managed by routine cleaning and trimming.

**Respiratory.** Cases of pneumonia, tracheitis, and bronchitis are seen, especially in old camp elephants and during the cold winter season. Signs include purulent trunk exudates, coughing, labored breathing, pain, fever, copious lacrimal secretion, and increased respiratory rate. Treatment includes parenteral broad-spectrum antibiotics and supportive therapy.

### Fungal Diseases

Cracked toenails infected with fungus have been observed. Regular cleaning and trimming of the affected area and application of a zinc-based ointment are instituted as treatment.

## PARASITIC DISEASES

### Intestinal Parasites

Endoparasites such as strongyles and gastric and intestinal nematodes are common (see Chapter 12 for details). Clinical signs may include a tendency to eat mud. Diagnosis is based on fecal floatation and observation of adult parasites in dung. Treatments used in Nepal include broad-spectrum oral anthelmintics such as fenbendazole (Panacur®, Intervet, India, Ltd.) and ivermectin. These medications are mixed in molasses and fed to elephants. Camp elephants are dewormed every 6 months. Ivermectin (Ivomec®, Glaxo-Smithkline Laboratories, India Ltd.), 0.1 mg/kg SQ (a total dose of 500 mg/adult) is used with good success. The injection site may show an initial local reaction (swelling), which normally subsides after a few days.

Severe submandibular and ventral abdominal edema has been observed in some elephants with liver flukes (*Fasciola jacksoni*). Treatment for fascioliasis is generally not instituted unless signs are severe. Triclabendazole (5–9 mg/kg) and oxyclozanide (6–8 mg/kg) have been used orally. Nitroxynil 34% (Trodax®, May and Baker, India) SQ was used in the past but produced a local tissue reaction and a fibrous growth at the injection site. Niclofolan (Bilivon®, Bayer-Germany) is also used against fascioliasis at 2 mg/kg SQ as a single injection. The incidence of fascioliasis and its treatment need further investigation. A large hydatid cyst (17 cm diameter) was found by the author on the liver during necropsy of a 25-year-old male elephant that died of unknown cause.

### External Parasites

The elephant louse, *Haematomyzus elephantis* causes pruritus, dry skin, and scale formation on the neck, earflap, ventral abdomen, axillary area, and around the base of the tail. Animals rub the affected part against trees or other hard objects. Frequent striking of the tail against the body is observed in severe cases. Lice occur on elephants that are not bathed regularly and when bathing protocols are not strictly followed. A commercial, organo-phosphorus–based agricultural antiparasitic dip (Neguvon®-Bayer, Germany) is used. Treatment also includes rubbing the affected areas with a brush, and applying an herbal ointment made of Indrabaron and Arjuna bark extracts (Himax®, IEL Limited, Ennore,

Madras, India). Ticks are seen occasionally, but they generally do not seem to be a health problem.

### Blood Parasites

Loss of general condition, depressed appetite, anemia, and hemoglobinemia may indicate blood parasites. An elephant with dark-colored urine, dullness, anorexia, weakness, and a body temperature of 37.5°C (99.6°F) was diagnosed with babesiosis. Transmission of babesiosis by various ticks, including *Boophilus annulatus, Bunicoplus* spp., *Ixodes* spp., and *Ripicephalus* species, have been reported.[1] Treatment with diminazine azeteurate (Berenil®, Intervet, India Ltd.) in two doses at 5 mg/kg given IM at an interval of 3 days was successful. Streptopenicillin (Dicrysticin® LD, Sarabhai Zydus, India Ltd.) comprised of 2.5 grams streptomycin sulfate, 150,000 IU procaine penicillin, and 500,000 IU penicillin G sodium/vial, was also given IM daily for 5 days. Symptoms resolved within 3 days.

## NONINFECTIOUS DISORDERS

### Nutritional

Elephants are given concentrate feed (rice, molasses, and common table salt) in addition to the free choice of seasonal fodder. Estimates of nutrient intake, based upon diet composition, have suggested that dietary concentrations of zinc and sodium may have been marginal, but the absence of signs of any nutrient deficiencies indicates that dietary husbandry in the camps was generally satisfactory.[5]

### Tusk and Dental

Tusk injuries with hemorrhage and pain are seen occasionally. Infection due to improper and too-short trimming of the tusk has been observed. Treatment includes sedation with xylazine hydrochloride, cleaning and irrigating the injured site with antiseptic solutions, and administration of parental broad-spectrum antibiotics. Antibiotic ointments with fly repellents (Himax®) are applied topically.

Old elephants lose their teeth and cannot eat normal elephant fodder and concentrate feed (rice paddy). These elephants are given selected grass and cooked husked rice with added molasses.

### Gastrointestinal

**Colic.** Signs of colic observed in camp elephants include frequent lying down and getting up, frequent urination, lifting the hindleg, and resting or leaning against walls or support/tethering polls. Biting the tip of the trunk with excruciating pain and vocalization has been observed in extreme cases. Elephants avoid even favorite food treats (fruit and molasses). Treatments include antispasmodics such as hyoscine butylbromide (0.04 mg/kg IV or 0.1 mg/kg IM), antihistamines such as chlorpheneramine maleate (0.2–0.5 mg/kg IM, Histal®, Vet Care Division, Tetragon Chemie Ltd., Bangalore, India), and antibiotics in chronic cases. Colic in baby elephants due to unhygienic milk preparation is also reported. The condition resolves with improvement in milk preparation.

**Impaction.** Cases of severe impaction of dry fibrous fecal material with dehydration are also seen. These elephants are restricted to concentrate feed for a few days. Such elephants generally recover well after passing impacted stool. Soap and water and/or mineral oil are used per rectum to promote defecation. Electrolytes (Rintose®) are given IV or rectally as supportive therapy.

**Nonspecific diarrhea.** This condition is seen occasionally and usually resolves with a change of fodder/grass and limiting the usual ration.

**Ocular.** Unilateral and bilateral cataracts are seen in some elephants. Working in the forest with the potential for eye injuries appears to be a contributing factor for cataract development. Elephants seem to behave normally unless there are complications from secondary infection. Recent corneal opacities have been cleared by dusting calomel into the cornea daily for more than a week.

**Reproductive.** Pregnant females have been observed in labor for up to 36 hours. Some delivered calves without medical intervention, although most of the calves were stillborn after such prolonged labor. In a few cases oxytocin (2000 IU IM) was given to aid expulsion of the fetus.

The Department of National Parks and Wildlife Conservation manages a successful breeding program at the Captive Breeding Center (see Figure 35.18).

***Figure 34.18.*** *Baby elephants are a common sight and an attraction at the Elephant Breeding Center in Chitwan (courtesy of Dr. Gretchen Kaufman).*

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# Section VIII—Sri Lanka

Indira Silva and Ashoka Dangolla

## INTRODUCTION

The elephant in Sri Lanka (*Elephas maximus maximus*) is a national treasure and is closely associated with culture, religion, society, and even politics (see Figure 35.19). It was declared a protected species in Sri Lanka in 1937 under the Fauna and Flora Protection ordinance No. 2.[26] The elephant is a "flagship species," whose conservation will result in the maintenance of biological diversity and ecological integrity in Sri Lanka.[10] The majestic presence of the elephant makes a colorful contribution to almost all Sri Lankan festivals. Elephants also play a vital role in the tourist industry, which is a leading foreign currency earner.

Ancient sinhala palm leaf manuscripts describing the veterinary aspects of elephant management are preserved in the museums in Colombo and Kandy. Important works are the Hasti Yoga Satakaya and Hasti Silpaya, which describe numerous conditions including eye and skin diseases; gastric, bowel, and bilious disorders; and the preparation of ointments, pastes, powders, pills, etc. for their treatment.[5]

Western therapy for elephants evolved later, and therefore, it is not surprising that elephant owners have a great deal of faith in native therapy. Native therapies have been passed down through generations but documentation is rare. Correct identification of herbs is one shortcoming of this practice. Based on the authors' experience, the use of native medicines may complement Western therapy in some instances; these are discussed below.

### Status of Elephant Populations

Elephants are broadly divided into two "races," based on morphological features: *Ruhunu gataya* (short and stout) in the southern region and *Vil aliya* (*Elephas maximus vi-*

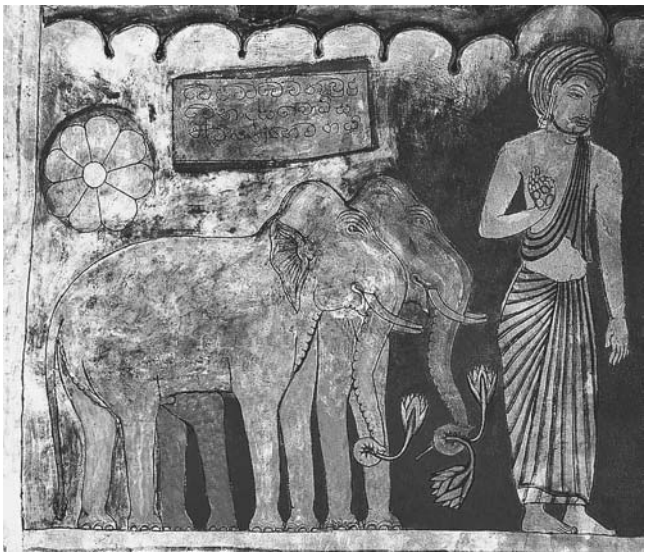

***Figure 35.19.*** *This 18th-century cave painting in the rock temple at Dambulla is one of many depicting elephants (photo courtesy of Hank Hammatt).*

*laliya)* (tall and elongated) in the mahaweli region.[9] Four types of male elephants are identified: tuskers (*Ātha*), single-tusked male (*Ek-Danteya*), tush elephant (*Aliya*), and without tushes (*Pussa*).[6] Only 7.3% of bulls are tuskers.[10]

**Wild elephants.** Elephants, with density estimates ranging from 0.12–0.64/Km$^2$ are found in several national parks. The estimated population of 4000 appears to be biased in favor of the adult animals, with a 1:3 male-to-female ratio.[10,14] Herds are composed of one or more adult females and their offspring. Groupings of

3–6 are common, and the largest group observed was 124. Increasing human population and conversion of forests for agricultural use have substantially reduced the land available to elephants.[26] Wild elephants confined to patches of forests surrounded by human settlements may be killed, injured, or orphaned by irate farmers or poachers.[10,26] The laws are relatively lax and poachers may receive only minor fines. Officials often have inadequate knowledge of the laws. Recently the Environmental Foundation Ltd. has helped evict illegal squatters from protected lands.

**Captive elephants.** The largest captive elephant herd in the world is at the Pinnawela Elephant Orphanage (website http://www/elephant.se). The orphanage began in 1975 with 5 babies and houses 65 elephants as of this writing. Providing refuge for injured, orphaned, or abandoned elephants was established[26] (see Figure 35.20). The orphanage plays an important role in ex-situ conservation by promoting captive breeding. Between 1984 and 2000, there were 18 births. The orphanage also provides a venue for public awareness/education and research. Common problems at Pinnawela include diarrhea, colic, and eye conditions.[26]

About 180–190 elephants are privately owned. They are required to be registered with the Department of Wildlife Conservation. The majority were captured prior to 1970; capture permits have not been issued for >30 years. Few calves have been born under domestication, and most elephants are >40 years old. There is no breeding incentive for owners and many sold their elephants after the Land Reform Act of 1972, which limited individual land ownership to 50 acres.[12] Owners find it increasingly difficult to maintain elephants due to scarcity of food, unavailability of veterinary services in most areas, and shortage of work for elephants due to mechanization of the timber industry.

Although a protected species, elephants are considered the same as any domestic animal and may be bought, sold, and cared for at the owner's discretion.[19] There is no legal protection for the domestic elephant from ill treatment by unkind mahouts or owners.[12] Animal Welfare legislation for all animals is currently being drafted. In 1999 the Captive Elephant Owners Association was formed to improve welfare of captive elephants.

## INFECTIOUS DISEASES

### Tuberculosis (TB)

Tuberculosis is common in humans in Sri Lanka. The first elephant clinical case was reported in 2002.[2] Diagnosis was based on culture of respiratory discharges and was confirmed at necropsy. The trunk wash procedure recommended in the U.S. has not proven practical in Sri Lanka; most elephants are difficult to train because they do not tolerate handling of their trunks.

***Figure 35.20.*** *Elephants in the river at the Pinnawela Elephant Orphanage (photo courtesy of Hank Hammatt).*

### Rabies

Rabies is an unusual occurrence and the first documented case was in 1999.[28] An 84-year-old female with a history of lethargy became unsteady, aggressive, and restless and had bilateral temporal gland secretions. Anorexia and trunk paralysis were seen on day 6, she fell several times, and appeared blind. Her condition deteriorated rapidly and she died on day 9. The diagnosis was confirmed by fluorescent antibody. The serum neutralizing antibody titer was 0.68 IU/ml (normal elephant serum is <0.04 IU/ml). Elephants are vaccinated using a killed product upon request of owners.

### Toxoplasmosis Seroconversion

Seroconversion for antibodies to *Toxoplasma gondii* using a direct modified agglutination test has been demonstrated in Sri Lankan elephants.[3] A significantly higher number of females were positive compared to males. Exposure may have been through fecal contamination from wild or domestic cats. No clinical disease has been associated.

### Parasitic Diseases

Helminth species reported include *Murshidia murshida, Murshidia falcifera, Murshidia longicaudata Quilonia renniei, Quilonia travancra, Equinubria sipunculiformis, Decrusia additictia, Parabronema smithii, Grammocephalus hybridatus, Choniangium epistomum, Amira pileata, Bathmostpmum sangeri, Cobboldia, Schistosomes,* and *Fasciola*.[7,17,22,23] Most anthelmintics such as pyrantel, mebendazole, albendazole, and febentel are effective against helminths. Presently, oxyclosanide is the only drug available for fascioliasis. External parasitism is not a clinical problem because of thorough bathing practices adopted by the mahouts. However, *Haematomyzus elephantis* louse infestations and *Amblyomma testudinarum* tick infestation do

occur.[18] Blood parasites (schistosomiasis, babesiosis, and filariasis) also occur and are discussed in other sections.

## NONINFECTIOUS DISORDERS

### Wounds

A recent study revealed that 25% of observed wounds were caused by the misuse of the ankus (goad) by unskilled keepers.[21] Leg chains were responsible for another 25%. Other causes were gunshots, prolonged recumbency, and traffic accidents.

The treatment for fresh, superficial wounds consists of lavaging with clean water followed by irrigation with an antiseptic solution (hydrogen peroxide, chlorhexadine, isopropyl alcohol, povidone iodine, tincture iodine, or potassium permanganate) and a topical wound dressing. Dressings consist of povidone iodine alone or a paste made of coumaphos, propoxur, and sulphanilamide (Negasunt®, Bayer HealthCare) and povidone iodine with or without zinc oxide. A parenteral antibiotic is given to old elephants. Native wound treatments also appear to be very effective. In an informal study conducted by the authors, wounds treated with a combination of native oils and Western dressings healed 10 days sooner than wounds treated with Western medications alone. This novel Wound Dressing Oil (WDO) consisted of a mixture of margosa oil, gingerly oil, sulphur powder, amukaha (turmeric), Negasunt®, copper sulphate, zinc oxide, zinc sulphate, and povidone iodine.

Sometimes, the temporal gland of male elephants becomes inflamed, it becomes infected, and/or the duct becomes blocked. The infected glands are treated as an abscess. When there is blockage, the infected ducts should be flushed with saline before applying medication. The elephant is sedated with 40–80 mg xylazine intravenously for this procedure.[15]

Photosensitization from phenothiazine derivatives, reported in India, has also been seen in Sri Lankan elephants sedated with xylazine and acepromazine. Such wounds heal slowly, sometimes taking 6 months. A mixture of talc, zinc oxide, emollients, and antibiotics is applied topically.

Examination and treatment of wounds in the mouth is difficult. Sedation or a mouth gag may be required. A bite made of *Caryota* (palm) fiber soaked in potassium permanganate solution may be given to the elephant to chew so that the solution will drain down to the space between the tooth and the socket.

Wounds on the trunk have been surgically treated, though most of the time the elephants did not keep the sutures intact. Long-acting antibiotic injections and supportive therapy is recommended. The appetite should be monitored because pain may prevent eating. Nonsteroidal antiinflammatory drugs, such as diclofenac sodium (1500 mg IM followed by 1250 mg orally every 12 hours for 3–5 days) or indomethacine (625 mg orally, every 12 hours for 3–5 days) may be used to relieve pain. Diclofenac or indomethacine may be initiated by injection, continued with oral formulations, and supplemented with aspirin (25 × 300 mg) tablets for 10 days.

**Abscesses.** Pyogenic membranes in organized abscesses may be cauterized by infusing with 5% silver nitrate, 5% formalin, or phenol after removing the pus and cell debris. This infusion must be neutralized within 3–5 minutes, by lavaging with normal saline, to prevent damage to adjacent tissue. Some elephants require sedation. Most owners prefer the native treatment because the procedure involves minor interventions with no bleeding. Therefore, we often use the herbal mixture Karam to remove the pyogenic membrane and follow with a healing mixture. Karam is prepared by grinding and boiling the ingredients of one of the following recipes:[21]

1. *Elephantopus* (Athadi) leaves, *Cassia Alata* (Ath thora) leaves, *Leucas* (Thumba) leaves with palmanikkam (copper sulphate)
2. *Croton tiglium* (Jaapala) leaves, *Plumbago indica* (Rathnithul) leaves with palmanikkam (copper sulphate)
3. Bark of the *Terminalia ariuna* (Kumbuk) tree, leaves of *Cassia Alata* (Ath thora), turmeric (Amukaha), and sinnakkaram (zinc chloride)

The Karam is applied for 3–5 days, after which the pyogenic membrane falls off or can be pulled out, with no bleeding. The remaining wound is treated using any dressing mentioned above. With Karam, the healing time is reduced by 5–8 weeks, compared to the usual 18–20 weeks. Therefore, this native therapy warrants attention because it is inexpensive and "client friendly" compared to Western therapy.

### Gastrointestinal Disorders

**Malnutrition syndrome.** Most logging elephants in Sri Lanka pull from a bite tied to the logs, thus causing damage to their teeth. Broken teeth may be the most common cause for the malnutrition syndrome in Sri Lankan elephants.[1] Many of the captive elephants have their last pair of molars in wear and cannot adequately chew the regular diet of leaves and stems of *Caryota urens* (Kitul) and leaves of *Coccus nucifera* (coconut) and *Artocarpus heterophylus* (Jak). Weakness, lethargy, loss of condition in spite of a voracious appetite, passing undigested green matter in feces, and constipation are signs. Delayed wound healing, joint and foot problems, and ventral edema may occur as clinical signs progress. Hypoglycemia (as low as 56 mg/dl) and hypoalbuminemia (as low as 0.95 g/dl) may be seen. Severe hypoalbuminemia is one cause of ventral edema. Clinical stability may be achieved through weekly administration of intravenous amino acid infusions (6 × 500 ml Amino-

plasmal®, B. Braun Melsungen AG, Germany), Astymin®-3 tablets (India Ltd.), and multivitamin injections. Monthly anabolic steroid injections (250 mg nandrolone decanoate) combined with an oral mixture of multivitamins, minerals, cereals and pulses ("kurrakkan," *Eleucine coracana L.*; sesame, *Sesamum indicum L.*), coconut poonac (a cattle concentrate feed made of coconut), and rice bran mixed with honey or jaggery (palm sugar), for several months, improves the prognosis. Strict rest is essential. The prognosis is poor if the teeth have loosened from the socket.

**Constipation, colic, and impaction.** Constipation may result from fibrous foods or improper chewing due to worn teeth or greedy eating after a long period of starvation. Treatment includes intravenous fluids, vitamin B supplementation, and forceful feeding of a purgative/laxative, such as Dulcolax (bisacodyl 30 tabs × 10 mg, every 12 hours for 3–5 days). Tablets are concealed in ripe bananas or pineapples. Repeated use of purgatives is done with care because it is difficult to rule out a complete intestinal obstruction, intestinal rupture, or volvulus. Bisacodyl rectal suppository (400 mg) may be applied as a paste on the rectal mucosa at 12–hour intervals.

The above therapy may be combined with parenteral 100 mg metochlopramide (Perinorm®, Ipca Laboratories Ltd, India), which speeds gastric emptying, thereby stimulating peristalsis and subsequently appetite. Oral or parenteral diclofenac sodium or indomethacin is used if colic signs are prominent. Nonsteroidal antiinflammatory drugs are used with extreme care, due to their well-documented side effects such as gastrointestinal bleeding. The authors' experience on the response to intramuscular 1500 mg diclofenac sodium, followed by 25–30 tablets (50 mg) twice daily for 2–3 days, was prompt in relation to relief from pain and reduction in inflammatory swelling.[13] Two out of 15 elephants developed transient loose motion, which stopped immediately upon withdrawal of the drug. Continuous exercise should be included in therapy because it helps stimulate the peristalsis. A parasympathomimetic, such as neostigmine, injection may be administered as the last resort if intestinal obstruction is ruled out as a possible cause of constipation.

Several traditional therapies are available for constipation. One common practice is to instill sap from the leaves of the Kudu Daula plant (*Neolitsea cassia*), which contain 3% plant saponins, into the rectum. The authors have tested this herb in the form of an enema made of crushed Kudu Daula leaves and water. The therapy is repeated every 12 hours. In 3 days peristaltic movements may be palpated rectally, and it is possible to remove feces manually. The saponins lubricate the rectal contents. Lukewarm soap water enemas also stimulate bowel movements. However, repeated soap water enemas should be done carefully because soap can remove natural lubricating secretions, further aggravating the constipation.

Another traditional therapy is to feed 1 kg each of tamarind (*Tamarindius indica l.* (Fabaceae) and magnesium sulfate (epsom salts). Because most elephants refuse to open their mouths when they suffer from a digestive disorder, this medication should be given before the appetite diminishes. Most elephants readily eat this because they adore the taste of tamarind! This mixture also helps loosen the fecal matter.

**Diarrhea.** Diarrhea due to salmonellosis responds well to a combination of ampicillin (6250 mg) and metronidazole (10 bottles × 500 ml (Tablets Ltd., Madras, India) and the response is faster if the initial dose is given intravenously. *Balandtidium coli* is common and responds well to sulfa-trimethoprim combination orally or parenterally.

## Occular Conditions

Conditions seen in domesticated elephants are corneal ulcers, kerato-conjunctivitis, traumatic injuries, cloudy anterior chamber, and cataract. Food material, such as blades of grass or sticks used by mahouts, may cause mechanical injuries. Corneal ulcers/corneal opacities respond rapidly to early treatment with antibiotic ointments such as gentamicin or chloramphenicol with or without steroids (dexamethasone/betamethasone) applied every 12 hours. If fungi are involved in corneal infections, selection of ointments is difficult. Some owners use native eye medications with antifungal properties. The vision of an elephant with corneal opacity was successfully corrected with placental extract (Placentrex®, Albert David, Kolkata, India) injected subdermally in the upper eyelid[27] (see Figs. 35.4 and 35.5).

Filariasis, though not proven, may play a causative role in corneal opacity. Corneal ulcers and opacities are more common during the dry seasons. It is believed that direct sunlight may irritate the cornea. An association of Acanthamoebae with a seasonally occurring corneal ulcer was recently documented.[4] Elephant owners believe that when eye conditions resolve, elephants develop foot ailments. This may appear so, because foot conditions occur mostly during the wet seasons, which immediately follow the dry seasons of the year.

Total lendectomy of one eye was successfully performed to correct blindness due to a mature cataract.[16]

## Pododermatitis and Arthritis

Pododermatitis is a serious health problem in captive elephants. A survey of 55 elephants revealed 62% with pododermatitis of at least one foot and 27% with both hindfeet affected.[8] Cracks in the nails or sole and inadequate foot care are contributory. Working elephants are more prone to develop pododermatitis from walking long distances on rough surfaces and tarred roads. Standing in dirty stables throughout the night, espe-

cially during the festival seasons, is another risk factor. Aged females are more likely to have their feet soiled with dribbled urine compared to males.

The causative organisms are a mixture of bacteria, fungi, and yeasts. The bacterial species isolated from lesions were *Streptococcus, Staphylococcus (S. aureus), Klebsiella, Proteus, Corynebacterium,* and *Escherichia coli. E. coli* was detected from the floor of one stable.[8] Pododermatitis may be life-threatening and may require intensive treatment for months or even years. Recovery time is longer in older animals 46–65 years, compared to <45-year-olds (5–8 weeks).

Many owners and keepers still have faith in native therapy for this foot condition. The general treatment consists of soaking the affected feet in a dilute formalin bath (5%–6%) for 10 minutes to harden the sole, followed by either soaking in $KMnO_4$ solution for a further 10 minutes or applying a mixture of 2% gentian violet, zinc oxide, and sulphur. Povidone iodine also may be added if fresh wounds are seen. Parenteral antibiotics are administered to prevent septicemia.

The authors tested the efficacy of a bark extract of Kumbuk (*Terminalia arjuna*) as treatment for a 0.5 cm deep, 10 cm long lesion on the foot pad of a 20-year-old female elephant. The extract was applied twice daily after cleaning the wound with water. The lesion healed completely in 2 weeks.

Arthritis is common among old captive elephants and often results from physical injuries to the joints caused by keepers. Generally, the treatment begins with intramuscular diclofenac sodium or indomethacin followed by oral therapy. Acute cases are given intramuscular antibiotics, usually a long-acting penicillin. Adverse reactions (hemorrhagic diarrhea and even death) to diclofenac sodium have been reported in India, but this has not been experienced in Sri Lanka.

### Hormonal Imbalance

An elephant with a chronic skin condition inspired investigations on serum thyroid levels in domesticated elephants. The $T_3$, $T_4$, and free $T_4$ levels measured in 10 males and 14 females were less than values reported from other countries.[20] Males had a predisposition for higher free $T_4$ levels, which is the active form of the hormone. Domesticated females tend to have low triglyceride (TG) levels, which may be a reason for their low breeding efficiency, because TG has an involvement in lactation. Domesticated males have higher levels of cholesterol compared to wild males. Interestingly, cholesterol and triglyceride levels are also low in Sri Lankan elephants compared to other countries.[25] This could be due to genetic or dietary differences, workload, and other stress factors.

### Respiratory Diseases

Tuberculosis has been described under infectious diseases, above. *Mycobacterium elephantis* and *Pseudomonas* spp. were recovered from lung abscesses in two Sri Lankan elephants, at necropsy.[24]

### Other

Lead is higher in domesticated elephants (24.1 ± 13.3 µmol/l) than free-ranging elephants (7.2 ± 2.8 µmol/l).[11] Although there were no related clinical signs, the higher levels in elephants living in urban areas are noteworthy. Water sources contaminated with petrol, batteries, discarded oil filters, and—most importantly—vehicle exhausts, have been suggested as possible sources of lead.

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# 36 Conservation

Simon Hedges

## INTRODUCTION

Asian elephants and African elephants are listed as Endangered and Vulnerable, respectively, in the 2004 IUCN (World Conservation Union) Red List of Threatened Species.[8,2] Elephants enjoy various degrees of legal protection in all range states, although sport hunting of elephants is permitted in a number of states.[120,2] Asian elephants were included in Appendix I of the Convention on International Trade in Endangered Species of Wild Fauna and Flora (CITES) in 1975. Listing in Appendix I effectively bans all international commercial trade in a species and its products (such as ivory) among Parties to the Convention. African elephants were included in CITES Appendix I in 1989, but the populations of the following range states have since been transferred back to Appendix II: Botswana, Namibia, South Africa, and Zimbabwe.[2] This down-listing means that some strictly controlled trade is allowed. In July 1999, an experimental one-off sale of ivory from Botswana, Namibia, and Zimbabwe to Japan was permitted, following compliance with a number of agreed conditions; another one-off sale from South Africa, Namibia, and Botswana was approved in 2002, but that sale has not yet taken place because a number of preconditions have not been fulfilled.[35,96]

There is much uncertainty about the total (global) population size of any elephant taxa, but it is generally believed that African elephants are about 8–17 times more abundant than Asian elephants: with perhaps 40,000–50,000 Asian elephants[134] (but see Blake and Hedges[22]) and 400,000–660,000 African elephants.[23] As seen below, this uncertainty has major implications for planning conservation strategies.

Asian elephants formerly ranged from West Asia along the Iranian coast into the Indian subcontinent, eastward into Southeast Asia, including Sumatra, Java, and Borneo, and into China at least as far as the Yangtze-Kiang. This was over 9 million km$^2$.[134] Asian elephants are now extinct in West Asia, Java, and most of China.[104,134,22] They still occur in isolated populations in 13 states, with a possible range area of 486,800 km$^2$.[134,22] Most populations are threatened by poaching, habitat loss and other conflicts with humans (especially those caused by elephants' crop raiding behaviour), and a loss of genetic viability resulting from small population size and isolation.[120,48,149,84,134,22,62]

The African elephant may have once inhabited most of the continent,[37] and it is presumed to have been widespread everywhere south of the Sahara.[125] They were exterminated from North Africa in the Middle Ages, largely as a result of the ivory trade and desertification, although the capture of large numbers of elephants for warfare and circuses played a role.[121,37] Today, the range of African elephants is fragmented as a result of increasing agricultural development, but they remain widespread in sub-Saharan Africa, occurring in 37 states.[23,2] The total area of the range now occupied by African elephants is approximately 5,346,000 km$^2$, but only 35% of this is considered "known range," with the remaining 65% percent classified as "possible range," thus again emphasizing the surprisingly little that is know about these animals.[23,22] This paucity of data is an important theme in any discussion of elephant conservation (see next section).

## WHAT DO WE REALLY KNOW ABOUT THE STATUS OF ELEPHANTS?

### The Status of African Elephants

Since the mid-1990s considerable effort and resources have gone into compiling an African Elephant Database and using it to produce regular updates of the continentwide status of elephants.[119,18,23] Blanc and colleagues[23] infer from the most recent data a continental population total of 400,000–660,000 elephants. Large gaps in our knowledge still remain. For example, there are no credible estimates for the continental population prior to the late 1970s. Furthermore, although elephant

population trends in the 20th century are believed to have differed considerably across the different African subregions, the African Elephant Database cannot be used to determine trends in elephant numbers at the continental level. As the compilers of the Database note: "the data presented . . . cannot give any indication of overall changes in elephant populations in the period between the two reports"[23] (but see Blanc and colleagues[24]).

Southern Africa has the largest known population on the continent. Elephant numbers were reported to be on the increase in Botswana, northern Mozambique, Namibia, South Africa, and Zimbabwe, but possibly declining in parts of Zambia. There was insufficient information on the situation in Angola, Malawi, and the rest of Mozambique.[23] The current troubled political situation in Zimbabwe may have reversed the trend there.

In Eastern Africa, the consensus is that elephant numbers reached a peak ("regional population maximum") in the late 1960s and early 1970s.[2] Although the populations in Kenya and Tanzania remain relatively healthy, the long-term viability of some populations, particularly those in Eritrea, Ethiopia, Rwanda, Somalia, and parts of Uganda may be in doubt. The status of elephants in the Sudan and Somalia was uncertain due to civil strife.[23] In West Africa, the elephant population collapsed before World War I (1914–1918) because of intense hunting for ivory. Populations have remained at low levels ever since and the major conservation challenges now relate to the management of small genetically isolated populations surrounded by growing human populations.[13,23,148,2]

In Central Africa, data on trends prior to 1977 are scarce,[23] but there is little doubt that elephant populations have declined since,[102,21,71] not the least because much of the ivory entering West African markets originated in Central Africa.[36] Unfortunately, our knowledge of the subregion's current elephant population size is the weakest and is largely based on 15-year-old data and guesses.[23,22] This is particularly worrying because Central Africa accounts for approximately 42% of the estimated continental range. The prevalence of unrest in Central Africa adds to the problem by making survey and monitoring work difficult or impossible in several areas.[23,2]

It is clear from this brief summary that data quality varies tremendously between the subregions. The African Elephant Database includes four categories of population estimate for each range state and subregion: *known, probable, possible, and speculative,* each with a rather complicated definition based on the quality of the data used to make the estimate.[23] In central Africa, only 16,450 elephants are "definitely" known to exist, and the "speculative" estimate is 82,563. By contrast, in eastern and southern Africa, "definite" is by far the dominant category. In West Africa, the two estimates are similar.

Clearly there is an urgent need for better information about elephant distribution and numbers, especially for elephants that dwell in the forests of West and Central Africa.[22] Barnes and colleagues[17] made this point over 10 years ago, adding that ignorance of basic elephant biology was one of the four major constraints to forest elephant conservation. They further suggested that "detailed surveys of elephant numbers and distribution should be made in each country and repeated at regular intervals to determine trends." Unfortunately, in 2005, 15 years after the last regionwide survey in central Africa, such a system is still not in place, although the elements are beginning to come together as a result of the CITES Monitoring the Illegal Killing of Elephants (MIKE) program.[22,21]

## The Status of Asian Elephants

Unfortunately, no equivalent of the African Elephant Database exists for Asian elephants. Surprisingly little is known about the status of Asian elephant populations: Duckworth and Hedges[48] concluded that there were insufficient data to estimate elephant population sizes in Indochina (Cambodia, Lao PDR, Vietnam, and Yunnan [China]); Sukumar[133,134] was able only to provide a general overview for the Indian subcontinent; and Hedges and colleagues[62] argue that there are no reliable elephant population estimates for Indonesia outside of one province in southern Sumatra, and no meaningful estimate of Indonesia's national elephant population can be made.

The oft-repeated global population "estimate" of about 30,000 to 40,000 or 50,000 Asian elephants is in reality no more than a crude guess, which has been accepted more or less unchanged for a quarter of a century despite major loss of Asian elephant habitat (Table 36.1). With few exceptions, all we really know about the status of Asian elephants is the location of some (probably most) populations, with in some cases a crude idea of relative abundance. For some large parts of the species range we do not even know where the populations are, or indeed if they are still extant[22,62] (Table 36.2). A major effort to address this ignorance is long overdue.

## Why Counting Elephants Matters

Obtaining a better understanding of rangewide status and trends is critical because inaccurate data leads to the misdirection of funds and overlooked conservation opportunities.[22] For example, how can scarce resources be allocated appropriately when so little is known about the status and distribution of Asian elephants? Should we concentrate on those few populations that we know without any doubt are large and thus likely viable over the long term? If so, that would restrict Asian elephant conservation activities to a handful of sites, most of which are in southern India. Large populations like that recently identified in Sumatra's Bukit Barisan Selatan National Park would be excluded because of guesses of the kind that say elephants are "present in small numbers."[22]

**Table 36.1.** Published Estimates of the Total (Global) Wild Asian Elephant Population*

| Population Estimates | | | Area of Elephant | | |
|---|---|---|---|---|---|
| Minimum | Probable | Maximum | Range ($km^2$) | Year | Source |
| 28,000 | — | 42,000 | — | 1978 | Olivier 1978 |
| 23,000 | — | 41,000 | — | 1982 | Shoshani and Eisenberg 1982 |
| 34,470 | — | 53,710 | — | 1990 | Santiapillai and Jackson 1990 |
| 34,390 | — | 56,045 | 436,230 | 1992 | Sukumar 1992 |
| 35,000 | — | 50,000 | — | 1995 | WWF 2002a, who note that the data are based on 1995 "rough estimates" and that several populations have experienced steep declines since that time |
| 34,594 | 42,705 | 50,998 identified as "speculative numbers" | — | 2000 | Kemf and Santiapillai 2000, who cite the IUCN/SSC Asian Elephant Specialist Group (AsESG) as the source of their data and warn that almost all figures are very approximate |
| 34,594 | — | 50,998 | — | 2000 | WWF 2002b, who cite the AsESG as their source and note that all figures are very approximate |
| 41,410 | — | 52,345 | 486,800 | 2003 | Sukumar 2003 |

*Modified from Blake and Hedges 2004.

**Table 36.2.** Status of Asian Elephant Populations in 2003*

| Country | Status | Country | Status |
|---|---|---|---|
| India | Distribution well known. Some valid population estimates, but majority of population estimates are less rigorous. No national population estimate possible. | Vietnam | Distribution of relic populations generally well known. Crude information on relative abundance available. No population estimates, but most recent guesses suggest fewer than 150 elephants remain. |
| Nepal | Distribution moderately well known, probably three main populations. No population estimates available, no national population estimate possible. | Malaysia | Distribution moderately well known on peninsular, well known in Sabah. Crude information on relative abundance on peninsula, with better data available for Sabah. Sabah's elephants shared with Indonesian Borneo. Few if any high-quality population estimates available, no national population estimate possible. |
| Bhutan | Distribution poorly known or published. Resident populations exist, but many elephants shared with India. No population estimates available, no national population estimate possible. | Indonesia | In the mid-1980s, 44 discrete elephant populations were known to exist in Sumatra's 8 provinces; 12 of these were in Lampung Province. In 2003, only 3 of Lampung's 12 populations were extant. An unknown number of Sumatra's other elephant populations remain. Distribution moderately well known on Kalimantan. Robust estimates for two Sumatran sites (Bukit Barisan Selatan and Way Kambas National Parks). Some crude information about relative abundance exists for other sites, but no national population estimate possible. |
| Bangladesh | No recent studies of distribution. No population estimates available, no national population estimate possible. | | |
| China | Distribution moderately well known. Crude information on relative abundance; no population estimates, no national population estimate possible. | | |
| Myanmar | Distribution moderately well known. Crude information on relative abundance. No population estimates, no national population estimate possible. | | |
| Thailand | Distribution well known. Crude information on relative abundance; no population estimates, no national population estimate possible. | | |
| Laos | Distribution well known. Crude information on relative abundance; no population estimates available, no national population estimate possible. | Sri Lanka | Distribution well known. Crude information about relative abundance; no population estimates available, no national population estimate possible. |
| Cambodia | Distribution moderately well known, some crude information on relative abundance; no population estimates available, no national population estimate possible. | | |

*Condensed from Blake and Hedges 2004.

It is essential that management objectives be clearly defined for both protected and unprotected areas of elephant range. Information on elephant distribution and abundance and trends in these parameters are needed to set appropriate goals and to monitor the effectiveness of management actions, as well as to inform local people and other stakeholders.[85,23,137] But conservation action must not wait on population surveys: Both are needed now and the conservation community needs to make advances on both fronts simultaneously. There are clear

priorities for action even in those areas of Central Africa and Southeast Asia where knowledge of the status of elephants is the weakest.

## HABITAT LOSS AND DEGRADATION

Approximately 80% of the African elephant's range lies outside protected areas.[2] This poses special challenges for wildlife authorities and wildlife managers.[72] Indeed, habitat loss has been identified as the factor that most threatens African elephant survival outside protected areas and is thought by some to be a bigger threat to elephants than the ivory trade.[69] The extent of habitat loss has varied across Africa, and Central African savannah elephant populations have probably been more influenced by hunting than by habitat loss.[1] West Africa has seen rampant deforestation, and elephants are now confined to a few isolated patches. By contrast, there is still much elephant habitat in Central African forests. However, many areas are now forestry concessions, and although selective logging may benefit elephant populations by creating secondary forest, the human activities associated with logging—particularly the bushmeat trade and human settlement—are important threats to elephant populations.[23]

It was thought that declines in elephant populations mirrored increases in human populations, i.e., that there was a negative linear relationship between rising human density and declining elephant density at a coarse (national or subcontinental) scale. Using observed elephant densities and human population data, Hoare and du Toit[69] tested this prediction for savannah elephants and their results did not support the linear model; elephants persisted up to a certain point and then precipitously declined. The sharpness of the decline suggests that elephants can coexist with people up to a certain threshold, but when that point is reached they go elsewhere, presumably to areas less disturbed by people and their agricultural activities. The threshold of human density beyond which elephant populations disappeared appeared to occur when agricultural land became spatially dominant over the natural woodland (savannah) that constitutes elephant habitat, or when the human population reached about 28 people per square mile. The value of this threshold hypothesis is that it will help planners distinguish areas where savannah elephants can be conserved from those where they cannot. Unfortunately, it is unlikely to apply to forest-dwelling African elephants because the ecological requirements of forest elephants and the patterns of human land use are different.

The greatest threats to the Asian elephant are also habitat loss and fragmentation.[120,133,84,62] Leimgruber and colleagues[84] mapped *wildlands,* defined as large, unfragmented, and undeveloped areas, and they found that these areas accounted for only 51% of Asian elephant's range in the 1990s. Furthermore, only 16% of unfragmented wildland and only 8% of the species' entire range was protected. Although these calculations were of necessity based on scarce and often low-quality data on elephant distribution (as discussed above), they are likely indicative of the actual situation. Leimgruber and colleagues argue that maintaining large unfragmented wildlands will be an essential tool for the long-term conservation of elephants in Asia. Although this is doubtless true if we are to maintain ecologically functioning populations of elephants that retain at least some of their evolutionary potential, it is not the whole story. Many of India's, Sri Lanka's, and Indonesia's elephant populations live in highly fragmented areas in close proximity to sizeable human populations. The price paid in these situations, however, is a high level of human-elephant conflict, which can lead to the extirpation of elephant populations.[133,84,62] Even if effective methods for reducing human-elephant conflict can be developed, direct interventionist management of elephant populations and their habitat will be needed, and current levels of knowledge about Asian elephant habitat requirements, dietary ecology, and population viability are inadequate for the task.[84]

## HUMAN-ELEPHANT CONFLICT

The term *human-elephant conflict* is used to describe a wide range of rather different problems involving humans and elephants. Since human-elephant conflict is a major problem for all elephant taxa, it is useful to consider what is meant by the term in more detail. Arguably human-elephant conflicts include competition for habitat and problems of overabundant populations, as well as the injuries, deaths, and crop losses that can occur when elephants enter agricultural lands. However, habitat-related problems are quite different from the problems caused by elephants raiding crops, and they have different solutions, so it does not seem helpful to label all these problems human-elephant conflict. Creating a national park to help reduce the impact of habitat loss on a region's elephant population is different from building an electric fence to keep elephants out of agricultural areas, even if the creation of the park might lead to local increases in crop raiding by elephants. Furthermore, the term human-elephant conflict has generally been used to refer to the problems arising from crop depredations by elephants, and it seems wise to restrict its use to such problems. This may seem like an issue of semantics, but until these distinctions are made, it is difficult to either 1) understand the history of elephant declines in many areas or 2) decide how to manage human-elephant conflict.

A failure to distinguish adequately between chronic or occasional crop raiding and acute conflict over habitat has been a characteristic of the debate over human-elephant conflict in some areas, as is illustrated by the following quotation from the report on the 1993

Sumatran Elephant Population and Habitat Viability Analysis (PHVA) Workshop: "As a result of the decline in the forest cover and increase in the human population growth, the elephant-human conflicts in Sumatra have escalated. In extreme cases, the [Indonesian authorities were] forced to capture chronic raiders and rogue elephants with the view to minimizing the human-elephant conflicts. This has lead to the establishment of a number of Elephant Training Centers across Sumatra."[142] However, far from being a response to "extreme cases," capturing wild elephants was routine government policy in Indonesia until the Asian financial crisis of 1997 reduced operational budgets. Furthermore, the majority of elephants captured in Sumatra's Lampung Province, for example, were caught not because they were "chronic raiders" or "rogue elephants," but because they occurred in Production Forests that were scheduled for conversion to agriculture and settlements.[62] The remainder of this section will, therefore, use the term *human-elephant conflict* to refer to crop depredation by elephants.

Human-elephant conflict is not a new problem, and crop depredation by elephants has been and remains widespread in both Africa and Asia. In Africa, precolonial and early 19th century historians describe areas where elephant crop depredations caused food shortages or forced people to relocate their settlements, and some authorities believe human-elephant conflict has been a problem since the beginning of agriculture[20,100,101] (but see Martyn[89]). In Asia, ancient Indian sources refer to conflict between elephants and agriculturalists as early as the 5th or 6th century B.C., and again both the precolonial and colonial era literature refer to crop depredations by Asian elephants.[134]

Despite the fact that habitat loss and hunting have reduced the geographical range and numbers of Asian and African elephants alike, human elephant conflict is apparently intensifying and is now identified as one of the most serious threats to elephants in both Asia and Africa.[64,65,72,12,73] The explanation of this seemingly paradoxical situation is that where elephants persist, they are often forced into close contact with people, and contemporary social conditions often lower people's tolerance of elephants.[100] Nevertheless, much uncertainty exists about the magnitude of the problem. Farmers often exaggerate crop losses caused by elephants in the hope of receiving compensation or assistance toward reducing the problem.[19,100,63,62] The damage caused by other species, especially rodents, primates, birds, or insects, is often greater than that caused by elephants.[100,63,62] However, because elephants are large dangerous animals that often injure or kill people during crop raiding incidents, the reactions they inspire are far stronger than those generated by smaller and less dangerous animals. As a consequence, people living in central African forests "fear and detest" elephants.[12] Similar examples could be listed for other parts of Africa and Asia. As Naughton and colleagues[100] note "This animosity is an ominous sign for future elephant survival, particularly given the trend toward decentralized wildlife management throughout Africa. Under current conditions, most local farmers would eliminate elephants from their environment if given the choice."

Even when such extreme reactions are not the case, people affected by elephant depredations typically demand protection or compensation from government authorities, and if these are not forthcoming they often retaliate by killing elephants or by facilitating access for poachers. People also express their frustrations by sabotaging conservation projects.[63]

It is clear that conservationists need to increase tolerance of elephants. This will require reducing crop depredations, which in turn will often require a better understanding of why elephants are raiding crops in the areas affected (e.g., Naughton and colleagues,[100] Hoare,[67] and Sukumar[134]). It is important to recognize that no single factor will explain human-elephant conflict across Africa or Asia. As Hoare notes, elephants and agriculture meet and mix in many different ways with varying consequences.[64] The development of data collection systems that describe site-specific characteristics and also contribute to a general understanding of human-elephant conflict have therefore been identified as a priority need.[108] The African Elephant Specialist Group's development of practical tools, including a standard Data Collection Protocol, is helping meet this need.[64,67,45]

Methods for reducing human-elephant conflict have been discussed in numerous reports from Africa and Asia (e.g., Seidensticker,[122] Hoare,[64] Wunder,[152] Hill and colleagues,[63] and Sukumar[134]). Osborn and Parker[106] divide these methods into passive and active methods. Passive methods attempt to limit the movement of elephants into agricultural areas and include the use of trenches, thorny branches, wooden or stone fences, and electric fencing. To these passive methods, we should probably add crop loss compensation and insurance schemes.[66] Active methods include the "drive them away" defenses traditionally used by farmers (e.g., chasing animals by banging on tins or drums, shouting, and throwing firecrackers or stones, or firing shots in the air to scare elephants). Other active methods are more novel—for example, Osborn and Rasmussen[107] tested a capsicum-based aerosol and found it effective for repelling elephants, but expensive. Active methods are used during raids, typically in crop fields at night. Other active methods include translocations (moving elephants) or removing elephants from the wild. The latter has been used extensively in Indonesia, with little success, and the large number of captive elephants now held in the so-called Elephant Training Centers has become a major animal welfare problem.[59,62]

Osborn and Parker[106] argue that most of the methods in use fail due to technical, logistical, and manage-

ment reasons. They suggest that human-elephant conflict persists because of four factors: problems inherent in the one-off "technical fix" approach, lack of farmer vigilance and participation, the habituation of elephants to any one method, and the high human and social costs of living with elephants and other wildlife.[106] The relative ineffectiveness and expense of most large-scale methods of reducing human-elephant conflict, including electric fencing and translocations, have led to increasing calls for the development of land use plans that address human-wildlife conflict and other conservation issues (e.g., Naughton and colleagues,[100] Barnes,[15] Hoare,[68] and Osborn and Parker[106]). In theory, addressing human-wildlife conflict at the planning stage could help prevent conflicts, although in many areas settlements are too well established for this approach. Moreover, it is now generally accepted that the resolution of human-elephant conflict will require small-scale, site-based, participatory approaches by farmers with an emphasis on crop selection, field layout, and inexpensive methods for deterring elephants.[139,63,106]

A key strategy in many places is the creation of buffer zones between agricultural areas and elephant habitat (including but not limited to protected areas). These buffers will help to establish a zone of "reduced attractiveness" between the crop fields and the elephants' habitat.[122,141,106] Osborn and Parker[106] argue that an optimal buffer zone should contain unpalatable crops grown adjacent to suboptimal elephant habitat: "The active management of a buffer zone with low-cost string fences, coupled with a vigorous deterrence regime, may instil recognition in elephants . . . that the fence demarcates a 'no-go' area."[106] This approach has met with some success in Gabon, Ghana, the Central African Republic, and Zimbabwe, and it appears that simple nonelectrified fences (sometimes coated in noxious chili-pepper–laced grease) can deter elephants.[106] Other modifications to traditional deterrence methods, including burning dried cattle dung mixed with chili peppers to produce a noxious smoke, have also shown promise.

The work of Osborn and Parker[105,106] has shown that increased farmer vigilance together with a new range of deterrents seems to reduce the damage caused by elephants, but they stress that convincing farmers that they can—and should—take responsibility for protecting their crops is the key to the success of this approach: "The central theme that emerges from examination of the failures of intervention is the need to decentralize responsibility for crop protection to the farmers. This represents a considerable shift in thinking, because farmers have historically depended on centralized . . . units to reduce this conflict."[106] Furthermore, the methods need to be financially and technologically within the capacities of the people implementing them.[72,105,106]

## ILLEGAL KILLING OF ELEPHANTS

The illegal killing of elephants (often referred to as *poaching*) occurs for a number of often interrelated reasons: to obtain ivory, meat, and other body parts; in retaliation for crop raiding or human injuries and deaths caused by elephants; for sport; or simply because the elephants are perceived to be in the wrong place. In addition to reducing elephant numbers, poaching can also lead to highly skewed age and sex ratios, which can have a serious impact on population dynamics. Elephants are highly social animals renowned for close relationships, and their young are reared within extended family groups. Poaching can fragment these patterns of social attachment by killing the matriarch and older female caretakers (allomothers) and deprive young males of critical socialization periods with older males. This breakdown in social networks can lead to hyperaggression and abnormally early musth cycles.[25,83,90]

In the 1970s and 1980s, an increase in the price of ivory led to severe episodes of poaching in many parts of Africa, starting in unprotected areas but also penetrating major national parks and reserves, and elephant populations declined substantially in many areas.[43,23] During this period attentions were focused on the poaching of savannah elephants, and there was a tendency to think that the forest elephants of the equatorial regions were relatively untouched (e.g., Anon[4]). However, in the late 1980s, a survey of six central African countries (Democratic Republic of Congo [DRC, formerly Zaire], Congo, Cameroon, Gabon, Central African Republic [CAR], and Equatorial Guinea) was implemented. The goal was to provide data on the distribution and abundance of forest elephants in central Africa and to assess the impact of the ivory trade.[11] The survey produced a number of important conclusions: 1) Central Africa probably contained close to one-third of Africa's elephants in 1989;[17] 2) poaching was widespread throughout the central African forest, particularly in DRC;[3,94] and 3) human activity, particularly poaching, was the major determinant of forest elephant distribution and abundance in central African forests.[16]

The 1989 moratorium on the international ivory trade helped reduce poaching, but large numbers of elephants continued (and continue) to be killed. The presence of largely unregulated domestic ivory markets in many countries continued to drive poaching (see below), as has the growing commercial trade in bushmeat.[51,131,23,21] Road construction associated with logging operations and mining facilitated access to formerly remote areas and more human settlements have resulted in higher levels of human-elephant conflict. These factors contribute to higher poaching rates (e.g., Lahm,[77] Auzel and Wilkie,[9] and Stein and BCTF[131]). Recent work has shown that poaching in Central and Eastern Africa is likely linked to the increased presence of illegal weapons and influxes of refugees due to wide-

spread political unrest in the region.[131] Widespread poverty means that illegal activities are worthwhile if the risks of capture and punishment are negligible,[82] which is the case in much of Africa. Elephant poaching and ivory trafficking remain attractive sources of income for rural people, traffickers, and dealers (e.g., Fay and Agnagna,[53] Courouble and colleagues,[36] Nishihara,[102] and Inogwabini[71]).

Poaching is a major threat to elephants in Asia too, although reliable estimates of the number of elephants killed and the quantities of ivory and other body parts collected and traded are scarce (e.g., Sukumar and colleagues[136] and Milliken[98]). It has been argued that poaching is a relatively minor threat to Asian elephants because some males and all females lack tusks.[39] The reality is that elephants are poached for a variety of other products (including meat and leather) in addition to ivory, and poaching is now acknowledged as a threat to the long-term survival of some Asian elephant populations (e.g., Kemf and Santiapillai[73] and Menon[92]). Moreover, poaching of elephants for ivory is a serious problem in some parts of Asia.[133,93] In Periyar Tiger Reserve in southern India, for example, ivory poaching has dramatically skewed adult sex ratios: Over the 20-year period from 1969 to 1989 the adult male:female sex ratio changed from 1:6 to 1:122.[31] Selective removal of tusker males has several implications: sex ratios obviously become highly female biased, genetic variation is reduced, and fecundity and recruitment may decline.[136,134] Poaching of elephants is also a major problem in other parts of Asia. Large-scale hunting of elephants for ivory, bushmeat, hides, and other products has reduced their populations significantly over a wide area from Myanmar to Indonesia.[93,48,73,86,92,149,62]

### Monitoring the Illegal Killing of Elephants (MIKE)

Given the significance of the threat posed by poaching to elephants in Africa and Asia, and the lack of good quality data on poaching rates and elephant population status, the 1997 decision by the Conference of the Parties (CoP) to CITES to establish a monitoring system across the entire range of African and Asian elephants would seem to be a major step in the right direction. This monitoring system, known as the Monitoring the Illegal Killing of Elephants (MIKE) program, is intended to inform and facilitate decision making by the Parties regarding the protected status of elephants and to provide a systematic and detailed assessment of the impact of the Parties' decisions to allow, restrict, or suspend trade in a particular species (and/or its parts and derivatives).

The aims were revised at the Eleventh CoP in 2000, and were broadened to include "establishing an information base to support the making of decisions on appropriate management, protection and enforcement needs" and "building capacity in range States." Specific aims include the following: 1) "To measure levels and trends in the illegal hunting of elephants," 2) "To determine changes in these trends over time," and 3) "To determine the factors causing such changes and to assess to what extent observed trends are related to CITES changes in listings or ivory trade resumptions" (www.cites.org/eng/prog/MIKE). These objectives are to be achieved through a site-based system of collecting data on elephant population trends, the incidence and patterns of illegal killing, and the effort and resources employed in detecting and preventing illegal hunting and trade.

In 2001, a pilot project at three sites in the central African forest block demonstrated that implementation of MIKE in forests was feasible, and a full-scale program involving 55 sites across Africa was initiated. Implementation in Asia was delayed due to political problems and a lack of funds, but has now begun.

The MIKE program is already proving its worth. For example, as a direct result of surveys conducted under the MIKE program, Central African governments now have a quantitative understanding of the distribution and abundance of elephants in some of their most important national parks. MIKE has also shown that poaching of elephants for the ivory trade is still a major problem in Central Africa's national park system, that in many areas this is increasingly associated with the trade in bushmeat as well as ivory, and that the species is losing geographic range even within national parks. These surveys have demonstrated the value of MIKE as a tool to improve the management and conservation of forest elephants.[131,21] Although there are a number of logistical and technical problems that need to be addressed as the program moves forward (e.g., Walsh and White,[143] Barnes,[14] Reeve and colleagues,[116] Hunter and Milliken,[70] Blake and Hedges,[22] and Blake[21]), these are being addressed. If the MIKE program meets the challenge of effectively monitoring the status of all elephant taxa, it will go a long way toward filling the information void that still exists across elephant range in much of Africa and Asia. Given the previous overwhelming reliance on guesses or inappropriate survey methods, it is encouraging to note that MIKE program surveys have to be conducted using standardized methodology that has been approved by the program's Technical Advisory Group. Indeed, one of the most beneficial outputs from the MIKE process has been improved methods for estimating elephant density from dung-count–based surveys.[61]

## THE IVORY TRADE

The ivory trade debate is one of the most intractable problems of international wildlife conservation,[52] and the question of whether there should be trade in ivory is a major source of disagreement between elephant range states. Although a number of compromises have been reached in the various Range State Dialogue Meetings and other fora, there is "no meeting of minds on ways to

address the issue."[23] Should a limited, well-regulated, and sustainable trade in ivory (and other elephant products) be allowed? Did the one-off sale of ivory from three southern African countries in 1999 lead to a rise in the price of ivory and stimulate poaching? Is the illegal ivory trade increasing? The answers to these and other important questions remain unclear.

One significant problem has been the lack of good data on the ivory trade, and consequently the debate has not always been particularly well informed. This situation improved significantly in 1992, when TRAFFIC (the wildlife trade monitoring network) created the Bad Ivory Database System (BIDS) to hold records of ivory seizures since 1989, and again in 1997 with the creation of the Elephant Trade Information System (ETIS). Managed by TRAFFIC, ETIS, together with the MIKE program, was mandated by CITES to measure and record levels and trends of illegal hunting and trade in ivory in elephant range states and in trade entrepots; to assess whether and to what extent trends are related to changes in the listing of elephant populations by CITES and/or the resumption of legal international trade in ivory; to establish an information base to support decision-making and protection needs; and to build capacity in range states (http://www.cites.org/eng/prog/ETIS/index.shtml). Unfortunately, although all CITES Parties are obliged to report elephant product seizures within 90 days of their occurrence, many states have failed to comply.[98]

Advocates of a regulated trade in ivory argue that it would provide financial incentives for governments to manage their elephant populations carefully; that revenues generated could help fund elephant conservation and antipoaching activities as well as support rural development for people living in or adjacent to elephant habitat; and that those people who live with elephants need to derive benefits from them to compensate for the destruction elephants can cause. A trade ban arguably removes peoples' incentive to preserve elephant populations and this will drive poaching.[126,10,138] A trade ban also penalizes those countries in southern Africa that have managed their elephant populations well and seen populations rise as a result.

During the debate before the 1989 ban and for a while after it came into force, it was suggested that a trade ban would lead to rising prices on the illegal market, thus providing an incentive for poachers to kill more elephants.[10] This did not happen, although the evidence is inconclusive for some countries.[46,29]

Economic models that assume ivory harvesting can be monitored and controlled such that "optimal management" is possible lead to the conclusion that a trade ban is counterproductive because states would maintain larger elephant populations if they were allowed to trade in their ivory.[28] However, current economic models are "ecologically simplistic" in that they often assume that elephant populations will grow until they reach the "carrying capacity" of their environment and then remain at that level. Such assumptions ignore the growing awareness of the dynamic nature of many ecosystems[56,26] (also see Hambler and colleagues[58] and Gillson and colleagues[57]). Such models do not adequately describe the trade in ivory, nor do they reflect the reality of elephant protection and monitoring in many states. Poor governance and corruption are endemic in many elephant range states.[129] Poaching is a real threat to elephant populations in many areas (see above) and protecting elephants is expensive. It is not clear that the ivory trade would provide sufficient revenues to protect the elephants and legal trade (even restricted) provides camouflage for illegal trade, thus providing a market for poached ivory.[40,74,27,26] This last point is perhaps the most important. Trading by one country can affect poaching or smuggling rates in another. Bulte and colleagues[26] describe this as "a potentially important but untested hypothesis." However, since the existence of unregulated domestic ivory markets is known to stimulate poaching in other countries, it seems reasonable to assume that a limited legal trade would also facilitate the "laundering" of illegal ivory and thus drive poaching.

International attention has increasingly turned to these domestic ivory markets in recent years. A 2003 report "More Ivory than Elephants: Domestic Ivory Markets in Three West African Countries" found that unregulated domestic markets in Nigeria, Ivory Coast, and Senegal were a major force driving elephant poaching in the Democratic Republic of Congo, Cameroon, the Central African Republic, and Gabon.[36] More generally, the most recent ETIS analysis demonstrated that the illegal trade in ivory is most directly correlated with the presence of large-scale, poorly regulated domestic ivory markets and these markets are the principal drivers of elephant poaching. An assessment of 22 ivory carving markets in Africa and Asia estimated that the ivory of between 4,800 and 12,000 elephants was needed each year to support annual production needs. Again, the majority of poaching appeared to be in Central Africa.[97]

These problems are by no means confined to Africa. A series of reports by TRAFFIC and Save The Elephants found that the ivory trade in a number of Asian countries depended largely on illegal sources of ivory, and that regulation of ivory trading was poor in most surveyed areas. Many countries with ivory-carving industries received illegal Asian ivory from neighboring countries, although it is not always clear whether the original source of the ivory was Africa or Asia.[99,5,75,103,123,151] Thailand was by far the largest ivory market in Southeast Asia, supplied mainly by illegal imports of raw ivory from Africa and tusks from Myanmar.[86] In East Asia, China was the largest illegal manufacturer and importer-exporter of ivory, most of it from African elephants.[87] India's domestic ivory market was not investigated by Martin and Stiles, possibly because it appears to

be moving deeper "underground."[95,6] The Indian Government has, nevertheless, admitted that elephant poaching is increasing nationally and it blames this increase on the downlisting of a number of African elephant populations. Others argue that the increase is being driven by demand and facilitated by poor management of the country's protected areas.[6] As Milliken[95] notes: "With the world's largest Asian Elephant population, understanding the ivory trade dynamics in India remains an urgent priority." Furthermore, enforcement of existing laws regarding elephant poaching and ivory trade in India needs to be carried out more rigorously[6] (see also Menon and colleagues[93]).

It is encouraging that significant attention was paid to domestic ivory markets at the Thirteenth Meeting of the Conference of the Parties to CITES (CoP13) in October 2004. An action plan was adopted for the control of trade in African elephant ivory. This plan requires all African range states to prohibit unregulated domestic sales of ivory, to instruct all law enforcement and border control agencies to act to stop illegal trade across international borders, and to engage in public awareness campaigns to publicize these prohibitions. The action plan also called on the CITES Secretariat "to monitor all domestic ivory markets outside Africa to ensure internal controls are adequate to comply with the relevant provisions . . . on Trade in elephant specimens. Priority should be given to China, Japan and Thailand." A full copy of the action plan endorsed by the African Elephant Range States Dialogue is available at http://www.cites.org/eng/cop/13/docs/E13-29-1A.pdf. Countries that fail to address their unregulated domestic ivory markets face possible sanctions under CITES, including suspension of all wildlife trade options. It remains to be seen whether this action plan will bring domestic ivory markets under control, thus preventing the illegal killing of thousands of elephants every year. Early signs are not promising.[7,88]

## MANAGEMENT OF OVERABUNDANT POPULATIONS

The management of overabundant populations is one of the most controversial issues in elephant conservation. It is almost solely an issue for African elephants, although Sukumar[134] suggests that it could become an issue in some parts of Asia. During the 1960s and 1970s there were heated debates about how to manage those elephant populations that were deemed to be too large, and specifically whether those populations should be culled. For example, in Eastern Africa the majority of protected areas reported rapid increases in elephant densities,[23] and pilot culling schemes were conducted within a number of national parks and reserves.[81,23]

The wave of poaching that devastated elephant populations in much of Africa in the 1970s and 1980s effectively ended the debate in many countries, although it continued to be an issue of concern in South Africa.[1] Nevertheless, culling has not been employed in the southern African region since Zimbabwe discontinued the practice in 1988 and South Africa in 1994 (although trophy hunting is actively practiced and produces significant revenues).[23] The issue has, however, returned in recent years as a decline in poaching in some countries has allowed elephant populations to increase and/or where elephants are compressed into small protected areas.

Novel management strategies are now being employed to deal with high elephant densities, but culling is still considered an option since—it is argued—culling methods have evolved to a point where trauma is minimized. Translocation has also become a common tool in the southern African region, and even intact families and large bulls can now be moved. Research into immunocontraception techniques has progressed to the point that these methods are now being seriously considered.[54,146] All methods have generated considerable controversy.

Those advancing a need to cull or otherwise reduce elephant densities typically argue that the creation of protected areas and a reduction in hunting allow elephant densities to reach artificially high levels, and at these high densities elephants damage and kill trees at rates that cannot be sustained. As a result, elephants convert woodland landscapes into grasslands and this has negative consequences for a host of other species as well as reducing the aesthetic appeal of the landscape. These changes in elephant habitat are likely detrimental to the elephants themselves and could result in large-scale mortality of elephants, depending on whether the new conditions provide sufficient food for them. Accepting these arguments leads to the conclusion that the ecologically rational (and some would add humane) approach is to reduce overabundant elephant populations to below the carrying capacity of their habitat and use the revenue generated (e.g., by selling meat and leather) for park management and rural development (for further discussion of these issues, see the following: Pienaar,[110] Laws,[80] Laws and colleagues,[81] Hanks,[60] Cumming and colleagues,[38] African Elephant Specialist Group,[1] Sukumar,[134] Whyte and Fayrer-Hosken[146]).

Those opposing culling or otherwise reducing elephant densities generally accept that high densities of elephants in savannah ecosystems can lead to short-term modification or loss of woodland, but they argue that the long-term effects are less clear because our understanding of ecosystem processes and long-term elephant population dynamics is too poor. It is possible that the changes in vegetation will be relatively short-term and cyclical if, for example, self regulation of the elephant populations occurs as a result of declining fertility and increased mortality due to a decline in food availability. Alternatively, fire or climatic factors may have been responsible for tree declines in some areas.

For further discussion of these issues see the following: Spinage,[130] Caughley,[30] Sinclair,[127,128] Dublin and colleagues,[47] Dublin,[44] and Sukumar.[134]

Much of the more recent debate over the need to reduce elephant populations has focused on the question of whether elephant populations need to be controlled in order to prevent the loss of biodiversity (e.g., Whyte[145] and Whyte and Fayrer-Hosken[146]). However, the evidence for local elephant-induced species extinctions is equivocal (e.g., Cumming and colleagues,[38] Gillson and Lindsey,[56] and Whyte[145]). Proponents of control argue for protection of biodiversity through management actions aimed at maintaining elephant numbers at what they see as a desirable level—a "biodiversity carrying capacity" to use the terminology of Gillson and Lindsay.[56] Such arguments seem to discount both the dynamic nature of ecosystems and the now rather long-established doubts about the utility of the carrying capacity concept in wildlife management. For further discussion of these issues see the following: Mcleod,[91] Gillson and Lindsey,[56] Bulte and colleagues,[26] Whyte,[145] Hambler and colleagues,[58] and Gillson and colleagues.[57] Ethical arguments for and against culling have also been—and remain—strongly contested (see, for example, Poole,[111] Whyte,[144] Leader-Williams and colleagues,[83] McComb and colleagues,[90] Bradshaw and colleagues,[25] and Whyte and Fayrer-Hosken[146]).

Although the debate will continue, it is clear that long-term studies on elephant population dynamics and impact on vegetation need to be conducted.

## THE ROLE OF CAPTIVE ELEPHANTS

First of all we should clarify our terms. Captive elephants are often referred to as *domestic elephants*. Some argue that this is a misnomer. As Kurt and Mar[76] and others have written, so-called domestic elephants are not domestic animals in the same way that dairy cattle, for example, are domestic animals. Perhaps the most important difference is that they are not selectively bred for particular characteristics, as is the case with cattle, sheep, or horses.[33,34,113] Indeed, most captive Asian elephants are wild-caught, and many of the elephants born in captivity in the range states were sired by wild males.[76] *Domestic elephants* should therefore be thought of as captive wild animals and treated as such (but see Lair[78] who makes a case for the use of *domesticated elephants*). Although this may seem like mere semantics, the reality is that what we call these elephants affects how we see them from the legal and moral viewpoints and thus affects their welfare. Finally, it can also affect our perception of their importance for elephant conservation.

Captive elephants are not a major issue for the conservation of African elephants. There are only about 700 captive *Loxodonta*, most of which are in western zoos,[32] and a wild population of approximately 400,000–660,000 animals.[23] By contrast, there are about 15,000 captive Asian elephants, mainly in the range states,[76] and perhaps 30,000–50,000 wild elephants.[134] This great disparity reflects the very different histories of the human-elephant relationship in Asia and Africa, and in particular the importance (until recently) of elephants as working animals in Asia, notably in India, Myanmar, and Thailand.[134]

Within the Asian range states there are elephant populations in timber camps that are reported to be self-sustaining or growing (e.g., Sukumar and colleagues,[135] and Taylor and Poole[140]), but historically captive populations have been a drain on wild populations and continue to be in some areas, notably Myanmar (e.g., Shepherd[123]). Although in Indonesia, large numbers of elephants are still removed from the wild and taken to Elephant Training Centers (see above; also see Hammatt and Fahrimal,[59] and Hedges and colleagues[62]).

Another serious concern is that ivory from captive elephants (e.g., from trimmed tusks) finds it way onto the markets. For example, Thai law allows trade in ivory from captive Asian elephants, and this loophole is being exploited by dealers. As a result, tens of thousands of ivory products of doubtful origin are continuously and openly for sale throughout Thailand.[97] Captive elephants and details of their tusks should therefore be registered with the appropriate government authorities to prevent such abuses. Indeed, the urgent need for all captive Asian elephant populations to be properly registered has long been recognized, not just to prevent the "laundering" of illegal ivory but to protect captive elephants from abuse and to facilitate better management.[79]

Captive elephant–related problems are not restricted to the range states—elephants in western zoos are in crisis. An imbalance in sex ratios, the emergence of new diseases, and poor reproductive success have compromised the long-term viability of these captive populations.[140,42,41,147,32,114] Such concerns clearly call into question the "safety net against extinction" justification that is sometimes advanced for maintaining captive populations. There are also serious concerns about the welfare of zoo elephants, and many of these welfare issues are intimately linked with the poor reproductive performance of the captive populations.[140,112,32] Not surprisingly, there have been calls for a halt to the breeding and importation of elephants until the factors responsible for poor welfare have been identified and remedied (e.g., Clubb and Mason[32]). Rees[114,115] suggests that there is no realistic prospect of establishing self-sustaining captive populations of Asian elephants in western zoos. He argues that it may be more productive, until they die out naturally, to use the existing animals in western zoos as ambassadors to raise money for in situ conservation in the range states. But are captive elephants, *Loxodonta* or *Elephas*, in the range states or in western zoos, of any benefit to elephant conservation beyond this role as ambassadors? I believe the answer is a clear yes.

Captive elephants have provided ready subjects for study, and many of these studies have provided valuable insights into the basic biology of *Loxodonta* and *Elephas* that have benefited wild elephants (see examples in Riddle and colleagues[118]). In a similar vein, captive elephants have allowed biologists and veterinarians to develop and practice techniques in ways that would be impossible with wild elephants. For example, the discovery of infrasonic communication in captive elephants has suggested new ways of counting forest elephants using their calls.[109] Experiments with captive elephants have allowed comparisons between DNA extracted from the blood and feces of the same animals, and these have shown that reliable noninvasive genotyping of individuals is possible.[55] Fecal DNA can therefore be used by biologists to answer important questions about the genetic viability of small elephant populations or to count elephants using the powerful techniques of capture-recapture statistics.[50]

In the range states, captive elephants have been used to chase crop-raiding wild elephants, to patrol protected areas, and for ecotourism such as elephant-back wildlife watching.[120] Wild-caught captive elephants have been used to study defecation rates of elephants foraging freely on natural diets in wild elephant habitat, and this information has been used to help biologists count wild elephants in forests by allowing them to convert estimates of dung abundance into estimates of elephant abundance.[62] Methods to determine the age of wild elephants from dung dimensions have also been developed using captive elephants held in camps in the range states.[117]

The existence of large numbers of captive wild-caught elephants in Asia raises the possibility that these animals could be returned to the wild, perhaps in areas where wild elephant populations have been lost. However, there are risks associated with such reintroductions. The potential introduction of diseases to wild populations is one consideration. Another is that elephants accustomed to being close to humans could become fearless crop raiders, and experimental releases are needed to evaluate the feasibility of such reintroductions (Sukumar[134]). I would also argue that reintroducing captive elephants is not currently a conservation priority for Asian elephants—protecting the remaining wild populations and their habitats is a much higher priority.

## PRIORITIES

In a recent paper, the author and Stephen Blake argued that the following are all needed as a matter of urgency for Asia: "(1) open acceptance of how little we really know about the status of elephants in Asia; (2) adoption of appropriate peer-reviewed survey and monitoring methods; (3) improved interagency cooperation and data sharing to effect a comprehensive review of conservation priorities for Asian elephants based on what we really know, not guesses; (4) immediate efforts directed at protecting known key populations; and (5) surveys to locate remaining large populations."[22] Number (4) can be further broken down into protection of key elephant populations from poaching, habitat loss and degradation, and the effects of human-elephant conflict. Implicit in the priorities listed above is the need for education, awareness building, and the development of national and local capabilities for protecting and managing elephants and their habitats, and for managing and reducing human-elephant conflict.

For African elephants, the African Elephant Specialist Group has identified all the following issues as being of equal priority for the conservation of the African elephant: 1) law enforcement, poaching, and the ivory trade; 2) habitat loss; 3) local overpopulation of elephants; 4) improved elephant surveys; and 5) human-elephant conflict.[1,65] Although much has been achieved it is clear that adequately addressing these priority issues will require funds on a scale far larger than that currently available for either African or Asian elephants. The challenge is for the range states and the international conservation community to mobilize adequate resources to ensure the conservation of these remarkable animals.[22,132]

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Appendix

# 1 Abbreviations Used in This Book

## U.S. CUSTOMARY

av = avoirdupois
ft = foot, feet
gal = gallon(s)
in = inch(s)
lb = pound(s)
ton = 2000 pounds
mi = mile(s)
oz = ounce(s)
sq = square
yd = yard(s)
ac = acre(s)
sp = species
spp = species (plural)
BW = body weight
wt = weight
IM = intramuscular
IV = intravenous
IA = intraarterial
SQ = subcutaneous
IP = intraperitoneal
PO = by mouth or orally
SID = once a day
bid = twice a day
tid = three times a day
q = every
qs = quantity sufficient
min = minute(s)
h, hr = hour(s)
d = day(s)
y, yr = year(s)
apoth = apothocaries' weight (pharmaceutical)

## METRIC

cc = cubic centimeter = ml
cm = centimeter
cu = cubic
g = gram(s)
ha = hectare(s)
kcal = kilocalorie(s)
kg = kilogram(s)
l = liter(s)
m = meter(s)
mEq = milliequivalent(s)
ml = milliliter(s) = cc
mg = milligram(s)
mm = millimeter(s)
m = meter(s)
t = metric ton(s) = 1000 kg
µl = microliter
dl = deciliter = 100 ml

Appendix

# 2 Measurement Conversion Tables

## LINEAR

1 millimeter = 0.039 inch
1 meter = 3.281 feet
1 meter = 1.094 yards
1 kilometer = 0.621 mile
1 inch = 25.4 millimeters
1 foot = 0.305 meters
1 yard = 0.914 meter
1 mile = 1.609 kilometers

## VOLUME

1 liter = 33.815 fluid ounces
1 fluid ounce = 29.573 milliliters
1 liter = 1.057 quarts
1 fluid ounce = 0.03 liters
1 liter = 0.264 gallons US
1 pint = 0.473 liters
1 quart = 0.946 liters
1 US gallon = 0.83 British Imperial gal = 3.785 liters
1 British Imperial gal = 4.545 liters = 1.2 US gallons
1 cup = 250 ml
1 tablespoon = 15 ml
1 teaspoon = 5 ml

## AREA

1 hectare = 0.004 square miles = 4046.86 sq meters
1 hectare = 107,639.1 square feet
1 hectare = (10,000 sq meters) = 2.47 acres
1 acre = (43,560 sq ft) = 0.405 hectare

## MASS

1 milligram = 1/60 grain (apoth)
1 grain (apoth) = 60 milligrams
1 gram = 0.035 ounce
1 ounce (av) = 28.35 grams
1 gram = 15.432 grains (apoth)
1 pound = 0.454 kilogram (454 grams)
1 kilogram = 2.205 pounds
1 ton (2000 lb) = 0.907 metric ton
1 metric ton (1000 kg) = 1.102 tons
1 mg/kg = 0.454 mg/lb
1 mg/lb = 2.2 mg/kg

## TEMPERATURE (degrees Celsius to degrees Fahrenheit)

| C | F | C | F | C | F |
|---|---|---|---|---|---|
| 25 | 77.1 | 37.0 | 98.6 | 39.2 | 102.6 |
| 26 | 78.8 | 37.1 | 98.8 | 39.4 | 102.9 |
| 27 | 80.6 | 37.2 | 99.0 | 39.6 | 103.3 |
| 28 | 82.4 | 37.3 | 99.1 | 39.8 | 103.6 |
| 29 | 84.2 | 37.4 | 99.3 | 40.0 | 104.0 |
| 30 | 86.0 | 37.5 | 99.5 | 40.2 | 104.4 |
| 31 | 87.8 | 37.6 | 99.7 | 40.4 | 104.7 |
| 32 | 90.6 | 37.7 | 99.9 | 40.6 | 105.1 |
| 33 | 91.4 | 37.8 | 100.0 | 40.8 | 105.4 |
| 34 | 93.2 | 37.9 | 100.2 | 41.0 | 105.8 |
| 35 | 95.0 | 38.0 | 100.4 | 41.5 | 106.7 |
| 36 | 96.8 | 38.1 | 100.6 | 42.0 | 107.6 |
| 36.1 | 96.9 | 38.2 | 100.8 | 42.5 | 108.5 |
| 36.2 | 97.2 | 38.3 | 100.9 | 43.0 | 109.4 |
| 36.3 | 97.3 | 38.4 | 101.2 | 44.0 | 111.2 |
| 36.4 | 97.5 | 38.5 | 101.3 | 46.0 | 114.8 |
| 36.5 | 97.7 | 38.6 | 101.5 | 47.0 | 116.7 |
| 36.6 | 97.9 | 38.7 | 101.7 | 48.0 | 118.4 |
| 36.7 | 98.1 | 38.8 | 101.8 | 49.0 | 120.2 |
| 36.8 | 98.2 | 38.9 | 102.1 | 50.0 | 122 |
| 36.9 | 98.4 | 39.0 | 102.2 | | |

### Temperature Formulas:

°C to °F = (°C × 1.8) + 32 = °F
°F to °C = (°F − 32) × .555 = °C

Appendix

# 3 Sources for Agents Used in Sedating, Tranquilizing, Immobilizing, and Anesthetizing Elephants

## (See Also Chapter 9)

1. Abbott Laboratories, 100 Abbott Park Rd., Abbott Park, Illinois 60064, USA
2. Abbott Laboratories Ltd., Abbott House, Norden Rd., Maidenhead, Berkshire, SL6 4XE, UK
3. Abbott Laboratories SA (Pty) Ltd., Box 1616, Johannesburg, Republic of South Africa (RSA)
4. Alembic Ltd., Alemic Rod, Valdora 390 003, Gujarat, India, infoal@alembic.co.in

4a. Allergan, Inc., 2525 Dupont Dr., Irvine, California 92623-9534, USA

4b. AstraZeneca Pharmaceuticals, 1800 Concord Pike, Wilmington, Delaware 19850-5437, USA

4c. Aventis Pharmaceuticals, 300 Somerset Corporate Blvd., Bridgewater, New Jersey 08807-2854, USA

5. BE Animal Health, India
6. ARKO Laboratories, Ltd., P.O. Box 400, Highway 69 North, Jewell, Iowa 50130, USA
7. Ayerst Laboratories (Akromed Products [Pty] Ltd.), Box 42, Isando, 1600 RSA
8. Aventis Pharmaceuticals, Maharashtra, 400 093 India
9. Bayer Corporation, P.O. Box 390, Shawnee Mission, Kansas 66201, USA
10. Bayer plc, Animal Health Business Group, Eastern Way, Bury St., Edmunds, Suffolk, IP32 7AH, UK
11. Bayer SA (Pty) Ltd., Veterinary Division, Box 143, Isando 1600 RSA
12. Bayer India Ltd. (IBC), India

12a. Baxter Pharmaceutical Products, 95 Spring St., New Providence, New Jersey 07974, USA

13. Boehringer Ingelheim, 2621 North Belt Highway, St. Joseph, Missouri 64506-2002, USA
14. Cadila Pharmaceutical Ltd., Arkhej-Dholka Rd., Bhat, Ahmedabad 382 210, India
15. Centaur Laboratories, Box 334, Isando, 1600 RSA
16. Ciba-Geigy (Pty) Ltd., Box 92, Isando, 1600 RSA
17. CIBA Vison (UK) Ltd., Flanders Rd., Hedge End, Southampton, SO30 2LG, UK
18. ELANCO Animal Health, 4 Parkwood, Suite 125, 500 East 96th St., Indianapolis, Indiana 46240-3733, USA

18a. Endo Pharmaceuticals, 220 Lake Dr., Newark, Delaware 19702, USA

19. EVSCO Pharmaceuticals, P.O. Box 685, Harding Highway, Buena, New Jersey 08310, USA
20. Farnam Companies, 301 W. Osborn Rd., Phoenix, Arizona 85013-3028, USA
21. Fision Pharmaceuticals (Pty) Ltd., Box 12084, Chloorkop 1624 RSA
22. Fort Dodge Animal Health, 800 5th St., NW, P.O. Box 518, Fort Dodge, Iowa 50501, USA
23. Fort Dodge Animal Health, Flanders Rd., Hedge End, Southampton, SO30 4QH, UK
24. Glaxo Wellcome UK, Stockley Park West, Uxbridge, Middlesex, UB11 1BT, UK
25. Glaxo SA (Pty) Ltd., Box 3388, Halfway House 1685 RSA
26. Glaxo SmithKline Ltd., India
27. Hoechst-Roussel, Vet., P.O. Box 4915, Independence Blvd., Warren, New Jersey 07059, USA
28. Hoechst Marrion Roussel, Ltd., Aventis Pharma Ltd., Aventis House, 50 Kings Hill Ave., Kings Hill, West Mailing, Kent, ME19 4AH, UK
29. Hoechst Animal Health, Box 457, Kempton Park, 1620 RSA
30. ICI SA (Pty) Ltd., Box 11270, Johannesburg, 2000 RSA
31. Indian Immunologicals Ltd., Hyderabad, India
32. Intas Pharmaceuticals (Neovet Intas Animal Health Care), www.intaspharma.com
33. Intervet, 405 State Street, P.O. Box 318, Millsboro, Delaware 19966, USA

33a. Janssen Pharmaceutical Products, 1125 Trenton-Harbourton Rd., Titusville, New Jersey 08560-0200, USA

34. Janssen Animal Health Division of Janssen-Cilag Ltd., P.O. Box 79 Saunderton, High Wycombe, Buckinghamshire, HP14 4HJ, UK
35. Janssen Pharmaceutical Animal Health, Private-Bag 9, Motortown, 2111 RSA
36. Jeps Pharma, PVT Ltd., C-207, Naraina IndL. Area, New Delhi 110 015, India
37. Key Pharmaceutical, A Division of Schering-Plough Animal Health, 1095 Morris Ave., Union, New Jersey 07083, USA
38. Krüger Med—Pharmaceuticals (Pty) Ltd., Private-Bag X037, Motortown, 2111 RSA
39. Lennon Ltd., 280 Kent Ave., Ferndale, 2160 RSA
40. Lloyd Laboratories, 604 West Thomas Ave., P.O. Box 86, Shenandoah, Iowa 51601, USA
41. Lundbeck SA (Pty) Ltd., Box 2357 Randburg, 2125 RSA
42. Lyka Laboratories Ltd., Ankeleshwar, Gujarat, India
43. Mallinckrodt Veterinary, Inc. (formerly Pitman Moore), 421 East Hawley St., Mundelein, Illinois 60060, USA
44. May Baker Animal Health SA (Pty) Ltd., Box 819, Halfway House 1685 RSA
45. Merck (Pty) Ltd., Box 1998, Halfway House, 1685 RSA
46. MSD (Merck) (UK) Merck Sharp & Dohme Ltd., Jertford Rd., Hoddesdon, Hertfordshire, EN11 9BU, UK
47. Merial, 3239 Satellite Blvd., Building 500, Duluth, Georgia 30096, USA
48. Merial, Sandringham House, 110 Sandringham Ave., Harlow Business Park, Harlow, Essex, CM19 5TG, UK
49. Millborrow Animal Health, Box 334, Isando, 1600 RSA

49a. Orion-Farmos, P.O. Box 65, FIN-02101, Espoo, Finland

49b. Ortho McNeil Pharmaceuticals, 1000 Route 202, Raritan, New Jersey 08869-0602, USA

50. Palmvet Services, Box 1135, Rynfield, 1514 RSA
51. Park-Davis & Co Ltd., Lambert Court, Chestnut Ave., Eastleigh, Hampshire, SO53 3ZQ, UK
52. Park Davis, Box 2743, Randburg, 2125 RSA
53. Pfizer Animal Health, 235 East 42nd St., New York, New York 10017, USA
54. Pfizer Ltd., Ramsgate Rd., Sandwich, Kent, CT13 9NJ, UK
55. Pfizer Laboratories SA (Pty) Ltd., Box 783720, Sandton, 2146 RSA
56. Pfizer Animal Health, Ankeleshwar, Gujarat, India

56a. Pharmaceutical Associates, 201 Delaware St., Greenville, South Carolina 29605, USA

56b. Pharmacia Animal Health, 7000 Portage Rd., Kalamazoo, Michigan 49001, USA

57. Pheniz SA (Pty) Ltd., Box 1825, Honeydew, 2040 RSA
58. Phoenix Pharmaceutical Inc., 4621 Easton Rd., P.O. Box 8068, St. Joseph, Missouri
59. Ranbaxy Laboratories Ltd., 19 Nehu Place, 110 019, New Delhi, India, seema.phuja@ranbaxy.com
60. Rhône-Poulenc Rorer Ltd., Aventis Pahara Ltd., Aventis House, 50 Kings Hill Ave., Kings Hill, West Mailing, Kent, ME19 4AH, UK
61. Rhône-Poulema, Box 819, Halfway House, 1685 RSA

61a. Roche Laboratories, 340 Kingsland St., Nutley, New Jersey 07110-0602, USA

62. Roche Products Ltd., 40 Broadwater Rd., Welwyn Garden City, Hertfordshire, AL7 3AY, UK
63. Roche Products (Pty) Ltd., Box 4589, Johannesburg, 2000 RSA

63a. Sanofi Synthelabo, 90 Park Ave., New York, New York 10016, USA

64. Sarabhai Zydus Animal Health Ltd., India
65. Schering-Plough Animal Health, 1095 Morris Ave., Union, New Jersey 07083, USA
66. Searle, P.O. Box 53, Lane End Rd., High Wycombe, Buckinghamshire, HP12 4HL, UK
67. Searle GD (Pty) Ltd., Box 11128, Johannesburg, 2000 RSA
68. Smith Kline Beecham Pharmaceuticals pfc, Mundells Welwyn Garden City, Hertforshire, AL7 1EY, UK
69. Smith Kline Beecham Animal Health, Private-Bag X56, Halfway House, 1685 RSA

69a. Solvay Pharmaceuticals, 901 Sawyer Rd., Marietta, Georgia 30062, USA

70. Twins Pharmaceutical Ltd., Box 200, Isando, 1600 RSA
71. Upjohn (Pty) Ltd., Box 246, Isando, 1600 RSA
72. Unichem Laboratories Ltd., www.unichemindia.com

72a. Vedco Inc., 5503 Corporate Dr., St. Joseph, Missouri 64507, USA

73. Vetsfarma, Police Lines Rd., Jalandhar 144 001 India, vets@jla.vsnl.net.in
74. Vetindia, India
75. Wellcome Environmental Health (Pty) Ltd., Box 677, Kempton Park, 1620 RSA
76. Wildlife Pharmaceuticals, 1512 Webster Court, Fort Collins, Colorado 80522-2026, USA
77. Wockhardt Laboratories Ltd., India
78. Wyeth-Ayerst Pharmaceuticals, 555 Lancaster Ave., St. Davids, Pennsylvania 19087, USA
79. ZooPharm, 3131 Grand Ave., Suite B, Laramie, Wyoming 82070, USA

Appendix

# 4 Toxicology Terms, Abbreviations, and Equivalents

## TERMS

ppm = parts per million = 1/1,000,000
ppb = parts per billion = 1/1,000,000,000
mg% = milligrams/100 ml = mg/dl
mEq/l = milliequivalents/liter
mg/l = milligrams/liter
µg/100ml = micrograms/100 milliliters
µg/kg = micrograms/kilograms
mg/kg = milligrams/kilogram

## EQUIVALENTS

1 ppm = 1 mg/kg = 1 mg/1,000,000 mg = 0.91 g/ton, = 0.0001%
Calculation: 1/1,000,000 = 0.000001. Then, ×100 = 0.0001%

0.001 ppm = 1 ppb = 0.0000001%
0.01 ppm = 10ppb = 0.000001%
0.1 ppm = 100 ppb = 0.00001%
1.0 ppm = 0.0001%
10 ppm = 0.001%
100 ppm = 0.01%
1000 ppm = 0.1%
10,000 ppm = 1.0%

To convert ppm to percentage, move the decimal point four places to the left. To convert percentage to ppm, move the decimal point four places to the right.

1 mg/g = 1 milligram/gram = 1000 ppm
1 mg/100 g = 10 ppm
1 µg/kg = 1,000,000,000 µg
1 µg/1,000,000 mg = 1 µg/1000 grams = 1 µg/kg
1 µg/g = 1 microgram /gram = 1 ppm
1 µg/ml = 1 microgram/ milliliter = 1 ppm

## LENGTH

1 cm = 0.3937 in
1 meter = 39.37 ft
1 micron = $1 \times 10^{-6}$ meters
1 micron = $1 \times 10^{-3}$ = 0.001 millimeters
1 angstrom = $1 \times 10^{-5}$ = 0.00001 microns

## AREA

1 acre = 43,560 square feet
1 acre = 4,047 square meters
1 hectare = 2.471 acres
1 hectare = 10,000 square meters
1 square mile = 1 section = 640 acres

## WEIGHT

1 grain = 64.8 milligrams
1 ounce = 28.35 grams
1 pound = 454 grams
1 ton = 2000 pounds
1 ton = 907.18 kilograms
1 metric ton = 1000 kilograms
1 metric ton = 2204.6 pounds
1 gram = 15.43 grains
1 kilogram = 2.205 pounds
1 microgram (mcg, µg) = 0.000,001 gram
1 nanogram (ng) = 0.000,000,001 gram
1 picogram (pg) = 0.000,000,000,001 gram

## VOLUME, FLUID

1 ounce = 29.57 milliliters
1 quart = 0.946 liters
1 U.S. gallon = 3.785 liters
1 gallon water = 8.345 pounds
1 cubic foot = 28.32 liters
1 acre-foot = $3.259 \times 10^5$ gallons
1 liter = 1.057 quarts
1 liter = 0.264 gallons
1 liter = 61.03 cubic inches
1 liter = 1000 milliliters
1 teaspoon = 5 milliliters
1 tablespoon = 15 milliliters

## VOLUME, DRY

1 bushel = 8 gallons
1 bushel = 4 pecks
1 bushel = 1.24 cubic feet
1 cubic foot 28.316 liters
1 cubic foot = 25.714 quarts
1 quart = 1.101 liters
1 cubic meter = 35.314 cubic feet
1 cubic inch = 16.387 milliliters

## MISCELLANEOUS

### Energy

1 calorie = 4.184 joules
1 kcal = 1000 small calories or 4184 joules
1 joule = 0.239 calorie
1 calorie = 1000 calories = 0.003968 BTU (British thermal units)
1BTU = 252 calories (gram) (at 15°C)

### Blood Levels

g% = g/dl = grams/100 milliliters
1 µKat/l (SI units) = 60 U/l (units per liter, conventional units)
1 µmol/l = 1 micromol/liter (SI units)
1 mmol/l = 1 millimol/liter (SI units)

Appendix

# 5 Elephant Vital Signs and Physiological Parameters

Compiled by Susan K. Mikota

| Parameter | Value | Reference |
|---|---|---|
| Height (meters) | | |
| • *Elephas maximus maximus* (Sri Lanka) | 2.0–.3.5 | Shoshani 1992 |
| • *Elephas maximus indicus* (Mainland) | 2.0–3.5 | |
| • *Elephas maximus sumatranus* (Sumatran) | 2.0–3.2 | |
| • *Loxodanta africana africana* (Bush) | 3.0–4.0 | |
| • *Loxodonta africana cyclotis* (Forest) | 2.0–3.0 | |
| Weight (kg) | | |
| • *Elephas maximus maximus* (Sri Lanka) | 2,000–5,500 | Shoshani 1992 |
| • *Elephas maximus indicus* (Mainland) | 2,000–5,500 | |
| • *Elephas maximus sumatranus* (Sumatran) | 2,000–4,000 | |
| • *Loxodanta africana africana* (Bush) | 4,000–7,000 | |
| • *Loxodonta africana cyclotis* (Forest) | 2,000–4,500 | |
| Life span (years) | 50–70 | Wallach 1983<br>Kingdon 1979 |
| Blood volume (% of body weight) | 3.5 (n = 1) | Shoshani 1982 |
| Chromosome number | 56 | Hungerford 1966<br>Sakthikumar 1990<br>Suwattana 2000 |
| Dental formula | I 1/0, C 0/0, PM 3/3, M 3/3<br>Total = 26 | Shoshani 1994 |
| Formula to estimate body weight (not accurate for young elephants) | Weight (kg) = 18.0 (heart girth in cm) − 3336 | Hile 1997 |
| Skin thickness (cm) | 1.9–3.2 | Kock 1993 |
| Vertebral formula | C-7, T-19, L-3, S-5, Cd–27; | Shoshani 1982 |
| Total # bones | 282 | |
| Rectal temperature | 36–37°C<br>97.5–99°F | Elder 1975<br>Kock 1993<br>Buss 1965<br>Brattstrom 1963 |
| Heart rate (beats per minute) | 25–30 (standing)<br>72–98 (lateral recumbency) | Kock 1993 |
| Respiratory rate (breaths per minute) | 4–12 | Schmitt 2003 |
| Trunk capacity (liters) adult Asian | 8.5 | Shoshani 1992 |
| Urine volume/24 hr (liters/day) | 25–53 | Dutta 2003<br>Cheeran 2002<br>Benedict 1936<br>Simon 1958, 1959 |
| Average volume/urination (liters) max | 5.5<br>10.58 | Benedict 1936 |
| Frequency of urination/24 hr | 5–10 | Benedict 1936<br>Simon 1959 |
| Fecal production (kg/day) | ~110 | Dutta 2003 |
| Defecations/day Asian | 12–20 | Cheeran 2002 |
| Boluses/defecation Asian | 5–8 | |
| Wt/bolus Asian (kg) | 1–2.5 | |

(*continued*)

| Parameter | Value | Reference |
|---|---|---|
| Defecation rate (h/defecation) African | 1.41–1.91 | Coe 1972 |
| Wt/defecation African adult (kg) | 10.36 | |
| Total dung/24 hr African, adult female | 100 kg | |
| Systolic blood pressure (standing) (mm Hg) | 178.6 ± 2.94 (n = 7 Asian and 8 African elephants) | Honeyman 1992 |
| Diastolic blood pressure (standing) (mm Hg) | 118.7 ± 3.10 (n = 7 Asian and 8 African elephants) | Honeyman 1992 |
| Mean blood pressure (standing) (mm Hg) (Note that blood pressure increases when elephants are in lateral recumbency.) | 144.6 ± 2.90 (n = 7 Asian and 8 African elephants) | Honeyman 1992 |
| Arterial $PO_2$ (standing) (mm Hg) | 96.2 ± 1.55 (n = 7 Asian and 8 African elephants) | Honeyman 1992 |

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Appendix

# 6 Glossary of Terms Associated with Elephant Feet

Compiled by Murray E. Fowler

**Abrasion (sole)**—Excessive wear of a segment of the sole. Usually caused by stereotypic behavior, such as turning continually in one direction or pawing with a foot.

**Abscess**—A localized collection of pus buried in tissues, organs, or confined spaces.

**Abscess, subsolar**—An abscess located beneath the sole (slipper).

**Amble (modified pace, single-foot)**—A four-beat gait in which the limbs on the same side of the body move forward at the same time, but the hindfoot hits the ground slightly before the forefoot. The amble is the medium-to-rapid gait of the elephant.

**Ankylosis (ankyloses,** *pl.)*—The fusion of a joint due to disease, injury, or surgery. May be partial, associated with pain, or complete (often without pain), but may cause a mechanical lameness.

**Ankus (hook, elephant hook, bull hook, goad)**—A metal prod and hook attached to a handle. The points on the prod and hook should be dull to prevent accidental tearing of the skin.

**Arthritis**—Inflammation of a joint and surrounding structures.

**Arthritis, rheumatoid**—A chronic disease primarily of joints and usually involving multiple joints, marked by inflammatory changes in the synovial membrane and articular structures. Elephants may develop this kind of arthritis.

**Arthritis, suppurative (exudative)**—Arthritis with an exudate within or surrounding a joint.

**Asphalt (blacktop, paving, bitumen, macadam)**—A mixture of tars and small gravel that is used for surfacing roads, enclosures, feeding areas, and stalls.

**Bitumen (asphalt, blacktop, macadam)**—A mixture of tars and small gravel that is used for surfacing roads, enclosures, feeding areas, and stalls.

**Black tracts**—Holes in the sole that are filled with a blackish exudate and that need to be cleaned to reveal healthy tissue when trimming a foot.

**Blacktop (asphalt, bitumen, macadam)**—A mixture of tars and small gravel that is used for surfacing roads, enclosures, feeding areas, and stalls.

**Blister (vesicle)**—A small accumulation of clear fluid between the layers of the skin caused by friction or by some viral disease process.

**Blood blister (hematoma)**—An accumulation of blood in a confined space, in a tissue, such as the sensitive fibrous tissue beneath the sole. Caused by capillary or vessel rupture.

**Bruise (contusion)**—An injury to the skin or the sole caused by a blow, but without a break in the skin or sole.

**Callus, interdigital**—A localized proliferation of the horny layer of the skin between toenails. May be caused by pressure, friction, or infection.

**Canker**—A condition characterized by a chronic, moist pododermatitis. Commonly manifested as a generalized infection at the skin and sole junction as a pododermatitis.

**Carpus**—The knee of a horse, or wrist of a human. In the elephant, the carpus is part of the foot of the forelimb.

**Cellulitis (phlegmon)**—An acute, diffuse, edematous suppurative inflammation of the deep subcutaneous tissue, often surrounding an abscess.

**Contusion (bruise)**—An injury to the skin or the sole caused by a blow, but without a break in the skin or sole.

**Corium**—The highly vascularized fibrous tissue that nourishes the skin, toenail, and sole. The corium also provides the fibrous connection between the nail and P-3 (distal phalanx).

**Coronary groove (band)**—The base of the hoof containing the germinal epithelium from which the hoof wall grows downward. May be used to describe the top of the nail in an elephant.

**Crack, sole** - Cracks in the keratinized layer of the sole (slipper, pad) of an elephant foot.

**Crack, toenail, deep**—A crack penetrating the full thickness of the nail, into the quick.

**Crack, toenail, vertical**—Either a superficial or deep crack that parallels the vertical axis of the nail. Such a crack may originate at the bottom or the top of the nail.

**Crack, toenail, superficial**—A crack that penetrates only the outer layers of the nail, not into the quick.

**Crack, transverse (horizontal)**—A defect in a toenail characterized by a crack across the long axis of the nail. May be initiated by an infection of the nail bed.

**Cracked heels**—Cracks in the skin near the rear of the foot.

**Cushion, digital**—A mass of fatty-fibroelastic tissue occupying the space behind the digits of the elephant foot.

**Cuticle (eponychium)**—The narrow band of epidermis that extends from the base of the nail wall onto the surface of the nail.

**Cuticle, overgrowth**—Excessive growth of the epithelium at the top of the nail (see *hangnail*).

**Decomposed granite (DG)**—A type of coarse soil, derived from granite rock, which may be compacted into a firm surface.

**Digit**—A finger or a toe.

**Digital cushion**—A mass of fatty-fibroelastic tissue occupying the space behind the digits of the elephant foot.

**Digitigrade**—Having feet shaped so that the toes, but not the heels, are on the ground (dogs, cats). Elephants are semidigitigrade in the forefeet, and semiplantigrade in the hindfeet.

**Discharge, purulent**—A discharge composed of pus.

**Dovetailing (feathering)**—Beveling the edges of a crack or groove to prevent impaction of dirt or feces in the crack.

**Eponychium (cuticle)**—The narrow band of epidermis (skin) that extends from the base of the nail wall onto the surface of the nail.

**Exudate**—A protein-rich fluid composed of serum, blood cells (primarily leukocytes), tissue cells, and/or cellular debris escaping from a wound or from the surface of a diseased tissue.

**Exudation**—The flow of an exudate from a wound.

**Feathering (dovetailing)**—Beveling the edges of a crack or groove to prevent impaction of dirt or feces in the crack.

**Fissure**—Any cleft or groove (normal or abnormal) on the surface of an organ, such as the skin or sole of an elephant foot.

**Foot, forefoot (manus)**—The distal segment of the forelimb, including the carpus, metacarpals, digits, tendons, ligaments, nerves, blood vessels, joints, skin, and nails.

**Foot, hindfoot (pes)**—The distal segment of the hindlimb, including the tarsus, metatarsals, tendons, ligaments, nerves, blood vessels, joints, skin, and nails.

**Foot rot**—A general term used to describe different types of infection of the foot.

**Gait, amble**—A four-beat gait in which the limbs on the same side of the body move forward at the same time, but the hindfoot hits the ground slightly before the forefoot. The amble is the medium to rapid gait of the elephant.

**Gait, walk**—A four-beat gait, in which there are always three feet on the ground at any one time. This is the slow gait of an elephant.

**Germinal epithelium**—The layer of cells at the base of the epidermis of the skin, sole, and toenail, which continues the growth of the structures.

**Goad (hook, ankus)**—A metal prod and hook attached to a handle. The points on the prod and hook should be dull to prevent accidental tearing of the skin.

**Granulation tissue**—The newly formed vascular tissue, normally produced in the healing of wounds of soft tissue.

**Gravel**—A horse term describing an infection that usually begins at the white line (junction of the hoof and sole); progresses dorsally, deep to the hoof wall; and usually breaks out (heads out) above the coronary band. As applied to an elephant foot, it describes an infection that invades the tissue just deep to the nail at the bottom of the foot and then migrates dorsally to the top of the nail bed. It was once thought that the infection was caused by the migration of a piece of gravel. In the horse, a piece of gravel may be present at the bottom of the tract, but it is now thought to be coincidental to the infection.

**Hangnail**—A small piece of skin hanging by one end at the side or base of a nail. A significant problem of elephant feet that are not properly cared for.

**Heel cracks**—Cracks in the skin near the rear of the foot.

**Hematoma (blood blister)**—An accumulation of blood in a confined space, in a tissue, such as the sensitive fibrous tissue beneath the sole. Caused by capillary or vessel rupture.

**Hoof**—The hard, horny covering of the feet of horses, cattle, sheep, swine, and wild ruminants, but not elephants.

**Hyperkeratosis**—Excessive production of keratin or horny tissue.

**Infection**—Invasion of a tissue or organ with bacteria, fungi, or viruses.

**Ingrown toenail**—Abnormal growth of a nail into the adjacent soft tissue of the nail bed.

**Interdigital callus (callosity)**—A localized proliferation of the horny layer of the skin between toenails. May be caused by pressure, friction, or infection.

**Keratinization**—The normal process of production of horny tissue on the surface of the sole or production of the outer cells of the skin.

**Laceration (cut, wound)**—A wound caused by a sharp object (glass, metal, teeth, jagged edge of wood).

**Laminitis (founder)**—An inflammation of the lamina (the attachment between the hoof and P-3). In contrast with other animals that have no lamina to connect the toenails to P-3, elephants have simple lamina; hence, they may develop laminitis.

**Macadam (asphalt, blacktop, paving, bitumen)**—A mixture of tars and small gravel that is used for surfacing roads, enclosures, feeding areas, and stalls.

**Maceration**—The softening and degeneration of the sole or skin of the foot caused by prolonged exposure to moisture and feces.

**Manus**—The distal segment of the forelimb including the carpus, metacarpals, digits, tendons, ligaments, nerves, blood vessels, joints, skin, and nails.

**Metacarpal bones**—The cannon bone of a horse. In elephants, the bones between the carpus and the digits of the forelimb.

**Metatarsal bones**—The hind cannon bone of a horse. In elephants, the bones between the tarsus (hock) and the digits.

**Nail bed**—The tissue upon which the nail is situated.

**Nailing**—Penetration of the sole (slipper, pad) by a sharp object (nail, screw, bolt, piece of glass, sliver). The penetration may be only within the keratinized tissue or go into the corium or digital cushion.

**Onychia (onychitis)**—Inflammation of the matrix of the nail, resulting in the loss of the nail.

**Osteoarthrosis, (degenerative joint disease [DJD])**—An arthritic condition characterized by degeneration of the articular cartilage, hypertrophy of bone at the margins, and changes in the synovial membrane.

**Osteomyelitis**—An inflammation of bone caused by pyogenic bacteria.

**Overgrowth, cuticle**—Excessive growth of the epithelium at the top of the nail.

**Overgrowth, nail**—Elongation of the nail caused by failure to wear the nail properly. The toenail may grow as much as one-quarter of an inch per month.

**Overgrowth, sole (slipper, pad)**—Excessive keratin on the sole caused by failure to wear the foot properly.

**Pad (sole, slipper)**—The bottom of the elephant foot, characterized by a flexible, keratinized layer overlying a germinal epithelium and corium (fibrous tissue).

**Paronychia (perionychia)**—Inflammation involving the folds of skin and tissue surrounding the nail.

**Pedestal (tub)**—A reinforced stand or platform upon which an elephant is trained to place a foot for trimming.

**Pedicure**—Professional care and treatment of the feet.

**Periople**—The layer of soft, light colored horn (keratinized), covering the outer aspect of the hoof or nail of ungulates. One of the functions of the periople is to protect the hoof or nail from moisture penetration.

**Phalanx (phalanges, *pl.*)**—The individual bones of the digits. P-1 = proximal phalanx, P-2 = middle phalanx, P-3 = distal phalanx. Not all digits in the elephant have three phalanges.

**Phalanx, P-3, fractured**—Characterized by multiple segments of P-3, seen on a radiograph. May be the result of trauma, osteomyelitis, decalcification, or general degeneration.

**Phlegmon (cellulitis)**—A spreading, diffuse inflammatory reaction to infection with small pockets of pus. The infection may be just beneath the skin or extend into muscles and other vital tissues.

**Pit**—A hole or cavity in the sole (slipper, pad). The pit may be superficial or extend into the quick (sensitive tissue). A pit may be hidden by an overgrowth of keratinized tissue.

**Plantigrade**—Foot structure that allows the animals to walk with the toes in a horizontal position (bears, humans). The hindfeet of elephants are semiplantigrade.

**Pocket**—A hollow space or an enclosed space.

**Pododermatitis**—Inflammation of the skin, nail, and associated structures of the foot.

**Puncture wound**—A penetration of the skin or sole (slipper, pad) by a sharp object (nail, screw, bolt, piece of glass, sliver).

**Pus**—A protein-rich fluid composed of blood cells (primarily leukocytes), tissue cells and/or cellular debris and bacteria, escaping from a wound or from the surface of a diseased tissue.

**Pustule**—A visible collection of pus within or beneath the epidermis, often in a hair follicle or sweat pore.

**Quick**—The sensitive tissue (supplied with nerves and blood vessels) deep to the sole or nail.

**Ridge**—A long, narrow proliferation of keratin in the sole of the foot. The pattern of ridges and grooves may produce a unique footprint of each elephant.

**Seedy toe**—A horse disease characterized by horny, honeycombed fungal growth between the hoof wall and P-3. May be present in elephants.

**Sesamoid bone**—A small bone embedded in a tendon or joint capsule at points of heavy pressure or acting like a pulley where a tendon changes direction. Elephants have a pair (or fused) sesamoid bones at the distal end of the metacarpal and metatarsal bones.

**Slipper (sole, pad)**—The bottom of the elephant foot, characterized by a flexible, keratinized layer overlying a germinal epithelium and corium (fibrous tissue).

**Sole abrasion**—Excessive wear of a segment of the sole. Usually caused by stereotypic behavior, such as turning continually in one direction or pawing with a foot.

**Sole, bruised (contused)**—Inflammation of the sensitive tissue beneath the sole. Evidence of a bruise may be localized over the area. Later, the spot may show reddening of the keratinized tissue from a deep hemorrhage.

**Sole (slipper, pad)**—The bottom of the elephant foot, characterized by a flexible, keratinized layer overlying a germinal epithelium and corium (fibrous tissue).

**Subsolar abscess**—An abscess located beneath the sole (slipper, pad).

**Substrate**—By definition, this is a layer of soil beneath the surface layer. In practical usage, it is the composition of the surface upon which an elephant walks. Types employed in elephant enclosures include decomposed granite, dirt, sand, gravel, asphalt (black top), concrete (rough or smooth).

**Synovitis (tendosynovitis)**—Inflammation of the tendon sheaths that surround tendons in the feet.

**Tarsus**—The hock of a horse or the heel and ankle of a human. In the elephant, the tarsus is part of the foot of the hindlimb.

**Thrush**—A horse term used to describe a fetid, grey-to-black discharge alongside the frog. Usually associated with degeneration of keratinized tissue. In elephants, it may describe the foul smelling, accumulated debris in cracks, crevices, or pockets in the sole.

**Toenail**—The cornified (keratinized) structure at the extremity of a digit.

**Toenail, ingrown**—Abnormal growth of a nail into adjacent soft tissue of the nail bed.

**Tract, black**—Holes in the sole that are filled with a blackish exudate, and which need to be explored and feathered to healthy tissue when trimming a foot.

**Tub (pedestal)**—A reinforced stand or platform upon which an elephant is trained to place a foot for trimming.

**Ulcer**—A local defect or excavation of the surface of the skin or sole, which is produced by the sloughing of inflammatory necrotic tissue.

**Ungulate**—The usual meaning is *hoofed mammals,* but animals such as tapirs, rhinos, and elephants that have more of a nail than a hoof are often included in the ungulate category.

**Vesicle (blister)**—A small accumulation of clear fluid between the layers of the skin caused by friction or by some viral diseases.

**Waist**—The narrowed circumference of the foot just above the nails.

**Wound, penetrating (puncture)**—A wound caused by a foreign body (nail, screw, bolt, piece of glass, sliver) that penetrates the skin or the sole of the foot.

Appendix

# 7 Weight Conversion Chart for Asian Elephants

The figures in the following chart have been calculated based on the formula: Weight in kg = 18.0 (Heart Girth in cm) − 3336. Note that this formula provides only an estimate of weight and has an overall average error of ±8%. The average error may be much higher for younger elephants, and this formula should be used cautiously in elephants under 13 years of age. This formula is not accurate for African elephants. It is preferable to obtain scale weights whenever possible.

For further details, see Hile, E.M., Hintz, H.F. and Hollis, N. 1997. Predicting body weight from body measurements in Asian elephants (*Elephas maximus*). J Zoo Wildl Med 28(4):424–427.

| Heart Girth (cm) | Weight (kg) | Weight (lb) | Heart Girth (cm) | Weight (kg) | Weight (lb) |
|---|---|---|---|---|---|
| 190 | 84 | 184.8 | 380 | 3504 | 7708.8 |
| 195 | 174 | 382.8 | 385 | 3594 | 7906.8 |
| 200 | 264 | 580.8 | 390 | 3684 | 8104.8 |
| 205 | 354 | 778.8 | 395 | 3774 | 8302.8 |
| 210 | 444 | 976.8 | 400 | 3864 | 8500.8 |
| 215 | 534 | 1174.8 | 405 | 3954 | 8698.8 |
| 220 | 624 | 1372.8 | 410 | 4044 | 8896.8 |
| 225 | 714 | 1570.8 | 415 | 4134 | 9094.8 |
| 230 | 804 | 1768.8 | 420 | 4224 | 9292.8 |
| 235 | 894 | 1966.8 | 425 | 4314 | 9490.8 |
| 240 | 984 | 2164.8 | 430 | 4404 | 9688.8 |
| 245 | 1074 | 2362.8 | 435 | 4494 | 9886.8 |
| 250 | 1164 | 2560.8 | 440 | 4584 | 10084.8 |
| 255 | 1254 | 2758.8 | 445 | 4674 | 10282.8 |
| 260 | 1344 | 2956.8 | 450 | 4764 | 10480.8 |
| 265 | 1434 | 3154.8 | 455 | 4854 | 10678.8 |
| 270 | 1524 | 3352.8 | 460 | 4944 | 10876.8 |
| 275 | 1614 | 3550.8 | 465 | 5034 | 11074.8 |
| 280 | 1704 | 3748.8 | 470 | 5124 | 11272.8 |
| 285 | 1794 | 3946.8 | 475 | 5214 | 11470.8 |
| 290 | 1884 | 4144.8 | 480 | 5304 | 11668.8 |
| 295 | 1974 | 4342.8 | 485 | 5394 | 11866.8 |
| 300 | 2064 | 4540.8 | 490 | 5484 | 12064.8 |
| 305 | 2154 | 4738.8 | 495 | 5574 | 12262.8 |
| 310 | 2244 | 4936.8 | 500 | 5664 | 12460.8 |
| 315 | 2334 | 5134.8 | 505 | 5754 | 12658.8 |
| 320 | 2424 | 5332.8 | 510 | 5844 | 12856.8 |
| 325 | 2514 | 5530.8 | 515 | 5934 | 13054.8 |
| 330 | 2604 | 5728.8 | 520 | 6024 | 13252.8 |
| 335 | 2694 | 5926.8 | 525 | 6114 | 13450.8 |
| 340 | 2784 | 6124.8 | 530 | 6204 | 13648.8 |
| 345 | 2874 | 6322.8 | 535 | 6294 | 13846.8 |
| 350 | 2964 | 6520.8 | 540 | 6384 | 14044.8 |
| 355 | 3054 | 6718.8 | 545 | 6474 | 14242.8 |
| 360 | 3144 | 6916.8 | 550 | 6564 | 14440.8 |
| 365 | 3234 | 7114.8 | 560 | 6744 | 14836.8 |
| 370 | 3324 | 7312.8 | 565 | 6834 | 15034.8 |
| 375 | 3414 | 7510.8 | 570 | 6924 | 15232.8 |

Appendix

# 8 Conversion Between Conventional and SI* Units, Hematology

| | | Multiply By | | |
|---|---|---|---|---|
| Analyte | Conventional Units | Conventional to SI | SI to Conventional | SI Units |
| Erythrocytes, RBC | $10^6/mm^3$ | $10^6$ | $10^{-6}$ | $10^{12}/l$ |
| PCV, hematocrit | % | 0.01 | 100 | l/l |
| Hemoglobin | gm/dl (gm%) | 10.0 | 0.1 | gm/l |
| MCV | $\mu^3$ | No change | No change | fl |
| MCHC | gm/dl (gm%) | 10 | 0.1 | gm/l |
| MCH | µg | No change | No change | pg |
| Leukocytes, WBC | $10^3/mm^3$ | $10^6$ | $10^{-6}$ | $10^9/l$ |
| Platelets | $10^3/mm^3$ | $10^6$ | $10^{-6}$ | $10^9/l$ |
| Protein, total | gm/dl | 10.0 | 0.1 | gm/l |
| Albumin | gm/dl | 10.0 | 0.1 | gm/l |
| Globulins | gm/dl | 10.0 | 0.1 | gm/l |

*Système International (SI).

Appendix

# 9 Conversion Between Conventional and SI* Units, Blood and Fluid Chemistry

| | | Multiply By | | |
|---|---|---|---|---|
| Analyte | Conventional Units | Conventional to SI | SI to Conventional | SI Units |
| Albumin | gm/dl | 144.9 | 0.007 | µmol/l |
| Albumin | gm/l | 14.49 | 0.069 | µmol/l |
| Ammonia | µg/dl | 0.59 | 1.69 | µmol/l |
| Bicarbonate | mEq/l | No change | No change | mmol/l |
| Bilirubin | mg/dl | 17.1 | 0.059 | µmol/l |
| Calcium (serum) | mg/dl | 0.25 | 4.0 | mmol/l |
| Calcium (urine) | mg/24 hrs | 0.025 | 40.0 | mmol/24 hrs |
| Carbon dioxide | mEq/l | 1.0 | 1.0 | mmol/l |
| Chloride | mEq/l | 1.0 | 1.0 | mmol/l |
| Cholesterol | mg/dl | 0.026 | 38.67 | mmol/l |
| Copper | µg/dl | 0.157 | 6.35 | µmol/l |
| Creatinine (serum) | mg/dl | 88.4 | 0.011 | µmol/l |
| Creatinine (urine) | gm/24 hrs | 8.84 | 0.113 | gm/24hrs |
| Enzymes | U/l | 0.017 | 60.0 | µkat/l |
| Fibrinogen | mg/dl | 0.01 | 100.0 | gm/l |
| Globulins | gm/dl | 10.0 | 0.1 | gm/l |
| Glucose | mg/dl | 0.056 | 18.0 | mmol/l |
| Iron | µg/dl | 0.179 | 5.58 | µmol/l |
| Iron-binding cap. | µg/dl | 0.048 | 20.7 | µmol/l |
| Lactate | mg/dl | 0.111 | 9.01 | mmol/l |
| Magnesium | mg/dl | 0.41 | 2.43 | mmol/l |
| Magnesium | mg/dl | 0.882 | 1.216 | mEq/l |
| Myoglobin | mg/dl | 0.585 | 1.71 | µmol/l |
| $pO^2$ | mmHg | 0.133 | 7.50 | kPa |
| Phosphate | mg/dl | 0.323 | 3.1 | mmol/l |
| Potassium | mEq/l | 1 | 1 | mmol/l |
| Protein, total | gm/dl | 10 | 0.1 | gm/l |
| Protein, CSF | mg/dl | 0.01 | 100 | gm/l |
| Protein, Urine | mg/24hrs | 0.01 | 100 | gm/24hrs |
| Sodium | mEq/l | 1 | 1 | mmol/l |
| Thyroxine $T_4$ | µg/dl | 12.87 | 0.777 | nmol/l |
| Triiodothyronine $T_3$ | ng/dl | 0.015 | 65.11 | nmol/l |
| Triglycerides | mg/dl | 0.011 | 88.5 | mmol/l |
| Urea nitrogen, UN | mg/dl | 0.357 | 2.81 | mmol/l |
| Uric acid | mg/dl | 59.48 | 0.017 | µmol/l |
| Zinc | µg/dl | 0.153 | 6.54 | µmol/l |

*Système International (SI).

## Appendix

# 10

**American Zoo and Aquarium Association**
**Standards For Elephant Management and Care**
**Adopted 21 March 2001, Updated 5 May 2003**

The following standards apply to the husbandry and management of both African (*Loxodonta africana*) and Asian (*Elephas maximus*) elephants in AZA accredited institutions, AZA related facilities, and nonmember participants in the AZA Elephant Species Survival Plan (SSP). The intelligence, strength, and social needs of these magnificent animals can pose many challenges for captive managers. Institutions desiring to hold elephants should therefore understand the substantial human, financial, and ethical commitments involved in appropriately maintaining these large and potentially dangerous species (Hutchins and Smith 1999). These standards have been developed to guide institutions that are planning and improving their elephant programs and are considered during the AZA accreditation process and nonmember SSP participant evaluation. The AZA Board of Directors has instructed the Accreditation Commission to immediately require written verification from AZA member institutions holding elephants, certifying that they are meeting the required standards (BOD 3/25/03).

The AZA Board of Directors believes that the Association performs a valuable role in the cooperative development of standards for zoo and aquarium animal management and care, which are designed to advance the collective mission of AZA and its members. The development of these standards and the adoption of them through the AZA accreditation process is what sets AZA members apart from roadside animal attractions. The Board understands that there will be differences of opinion as to what constitutes appropriate standards. Standards evolve over time reflecting changes in knowledge, expertise, and public perception.

The AZA Board of Directors has asked the AZA Elephant SSP/TAG to begin formulating a draft vision for the future of elephant management in AZA accredited zoos. Because current standards are expected to change over time, it is recommended that members seeking to plan new elephant exhibits/care programs look to the vision, rather than the current standards, for guidance on where to go in the future.

Compliance with some minimum housing (specifically space, enclosure design, and elephant restraint device (ERD) requirements) must be implemented no later than five years from the issuance of these standards (1 May 2006). Institutions must have written implementation plans for compliance with these standards no later than three years from their issuance (1 May 2004). AZA accredited and related facilities must meet all other provisions described here within one year (1 May 2002) of the issuance of these standards, unless the Accreditation Commission approves a variance. Failure to meet basic AZA standards for elephant management and care will be noted during accreditation inspections. Current nonmember participants in the SSP will be given the same time schedule for compliance, but new nonmember participants must meet all new standards prior to approval.

Highlighted sections are recommendations or standards for which variances may be obtained.

**1. Abiotic Environmental Variables**

1.1. *Temperature*

1.1.1. Elephants must be kept outside on natural substrates as much as possible. Institutions should consider designing exhibits that allow elephants outdoor access twenty-four hours a day—weather, health, and safety permitting. During daylight hours, elephants kept outdoors can tolerate moderate temperature extremes. Provisions must be made to protect animals from adverse weather, including intense sunlight, chilling rain, sleet, etc. Animals kept outdoors must be monitored frequently at temper-

atures below 40 degrees F (4.4 degrees C). Facilities may install outdoor heat sources to extend the amount of time the animals are able to remain outside.

1.1.2. While outdoors, all elephants must have access to shade during daylight hours in temperatures above 80 degrees F (27 degrees C) and when they are exposed to direct sunlight.

1.1.3. Indoor holding areas must be ventilated, and heated to a minimum temperature of at least 55 degrees F (12.8 degrees C) at all times of the year. One room must be capable of maintaining a temperature of at least 70 degrees F (21.1 degrees C) and be free of drafts, for accommodating sick or debilitated animals.

1.2. *Humidity*—There are no standards for humidity at this time. Information is limited, but this does not seem to be of major concern for elephant management.

1.3. *Illumination*

1.3.1. Natural daylight cycles are adequate for elephants, even in temperate regions. Indoor areas must be well illuminated during daylight hours, followed by a period of darkness. Fluorescent lighting provides a sufficient spectrum of illumination; skylights, in addition to interior lighting, are highly recommended. Ample interior lighting must be available, as it is especially important to maintain staff safety.

1.4. *Space*

1.4.1. Indoor space must provide adequate room for animals to move about and lie down without restriction. A minimum of 400 sq. ft (37.2 sq. m) is required for a single animal, approximately 800 sq. ft (74.3 sq. m) for two animals, and so on (AZA 1997). Because of their size and space requirements, bulls or cows with calves must have a minimum of at least 600 sq. ft (55.7 sq. m) (AZA 1997).

1.4.2. Outdoor yards must have at least 1,800 sq. ft (167.2 sq. m) for a single adult individual and an additional 900 sq. ft (83.6 sq. m) must be added for each additional animal (AZA 1997). If this space is the only location for exercise, then it is recommended that the space per elephant should be even greater.

**Note: Institutions can petition for a variance from the current minimum indoor or outdoor space standards. The applicant must explain why their facilities are adequate, even though they do not meet the minimum size standard. Accreditation inspectors will take a holistic approach to accreditation inspections, rather than focusing on specific size measurements. Context is particularly important. For example, it may not be a problem that the indoor space requirements are under the standard by a small amount if a zoo is located in a warmer climate and the animals are outside most of the time. If, however, the zoo is located in a cooler climate and the animals are kept inside for many months during the winter, then the indoor space requirements must be met or, preferably, exceeded. Environmental enrichment programs should also be taken into consideration when evaluating space available.

1.4.3. Mature animals can reach a vertical height of 20 ft (6.1 m). Consideration of this must be given with regard to ceiling heights and fixtures (e.g., lights, heating units, plumbing, etc.) so that animals do not harm themselves or the facility.

1.4.4. All facilities must have the ability to separate and isolate animals to address behavioral concerns or allow veterinary procedures to occur (EMA 1999).

1.4.5. Outdoor yard surfaces must consist primarily of natural substrates (e.g., soil, sand, grass) that provide good drainage and have a cleanable, dry area for feeding (EMA 1999).

1.4.6. While outdoors, elephants must have access to sand or soil at all times for dust bathing (EMA 1999).

1.4.7. Rocks, tree stumps, or large sturdy objects must be provided in the exhibit so that the animals may use them for rubbing and scratching.

1.4.8. Elephant containment barriers must be in good condition and able to prevent elephant escapes. A wide variety of building materials can be used as long as they are able to withstand the animals' strength, contain the elephant in a specific space, and prohibit direct contact between elephants and the public.

1.4.9. Door and gate design is extremely important to ensure the safety of both elephants and keeper staff. Both doors and gates must be engineered to withstand extreme force. If mechanical opening devices, such as hydraulic or electrically powered drives are used, they must be able to be operated manually or with a backup generator in the case of a power failure.

1.4.10. Enclosures must be cleaned of excrement daily. Frequent daily manure removal is recommended and may be necessary for the maintenance of both sanitary and esthetic conditions (EMA 1999).

1.4.11. If the AZA Elephant SSP-managed population is to become sustainable, it is necessary to create housing for many more adult males (Wiese 2000, Wiese and Olson 2000). All institutions considering new construction for elephants should include holding space for adult males. Institutions modifying existing facilities should also make provisions for bull housing.

1.4.12. There are no standards on the visual, acoustic, and olfactory needs of elephants at this time.

1.4.13. There are no specific standards for the transportation of elephants at this time, but see Fowler (1995).

1.5. *Water and Moats*

1.5.1. While outdoors and weather permitting, elephants must have regular access to a water source, such as a pool, waterfall, misters/sprinklers, or wallow that provides enrichment and allows the animals to cool and/or bathe themselves.

1.5.2. Standing water in indoor floor areas can cause foot problems and become a breeding ground for bacteria. Floors must therefore be impervious to water, quick to dry, and sloped to a drain. Floor surfaces must be relatively smooth, but not enough so that they become slippery when wet. Conversely, very rough surfaces may cause excessive wear or irritate footpads.

1.5.3. Dry moats can pose a substantial threat to elephants and their use must be limited with the ultimate goal that they are eventually phased out. Moats that are deep, narrow-sided, and hard-bottomed can be particularly dangerous. Although there should be no risk of animals falling or being pushed into the moat, written animal extraction protocols must be in place for any moat that is more than 3 ft (1 m) deep, less than 10 ft (3 m) wide, and/or hard-bottomed.

**2. Biotic Variables**

2.1. *Food and Water*

2.1.1.Elephants must have access to clean, fresh drinking water (EMA 1999). When water containers are used, drinking water must be cleaned and refreshed at least twice a day. Containers must also be cleaned daily.

2.1.2.Fresh browse and produce should be used as dietary supplements and enrichment for the animals.

2.2. *Group Composition*

2.2.1. The minimum age offspring must remain with their mothers is three years. Some flexibility is necessary in cases of maternal rejection and when infants cannot be reestablished in their social group.

2.2.2. Institutions must have the ability to manage social compatibility as well as dominance and aggression among an elephant group (EMA 1999).

2.2.3. Institutions must have the ability to manage introductions and separations of a new female to a herd and, if the institution is a breeding facility, females to males for breeding, newborn calf to its mother, and calf and mother to the herd.

2.2.4. Institutions must provide an opportunity for each elephant to exercise and interact socially with other elephants (Taylor and Poole 1998, EMA 1999).

2.2.5. Adult males (six years and above) may be housed alone, but not in complete isolation (opportunities for tactile, olfactory, visual, and/or auditory interaction with other elephants must be provided) (Rasmussen et al. 1982).

2.2.6. A behavioral profile must be maintained for each individual elephant and updated annually.

2.2.7. All holding institutions must have a written environmental enrichment plan for their elephants and show evidence of implementation (Shepherdson et al. 1998, EMA, 1999, Shepherdson 1999).

2.2.8. Staff must be aware of each animal's social compatibility and the dominance hierarchies of the herd (EMA 1999).

2.3. *Group Size*

2.3.1. Zoos should make every effort to maintain elephants in social groupings. It is inappropriate to keep highly social female elephants singly (see Sukumar 1992, Taylor and Poole 1998, EMA 1999). Institutions should strive to hold no less than three female elephants wherever possible. All new exhibits and major renovations must have the capacity to hold three or more female elephants.

**Note: It is understood that obtaining additional elephants for zoo exhibits can be difficult at this time. Temporary variances will therefore be considered regarding group size requirements. Institutions that do not currently meet the group size standard should demonstrate that they have requested assistance from the SSP in obtaining additional animals.

It is recognized that some socially aberrant adult females currently exist and these elephants can be managed singly if the institution has made every effort to introduce them to a social group and the SSP agrees that the antisocial behavior is not correctable.

2.4. *Human-animal Interactions*—A minimum of two qualified elephant keepers must be present during any contact with elephants. A qualified keeper is a person the institution acknowledges as a trained, responsible individual, capable of and specifically experienced in the training and care of elephants.

2.5. *Introductions*—There are no specific standards for elephant introductions at this time, but see Lindburg and Robinson (1986) and Krantz (1996).

**3. Health and Nutrition**

3.1. *Diet*

3.1.1. High quality and nutritionally correct food must be provided in sufficient quantities to maintain animal health and appropriate weight (EMA 1999). Hay and grain should be formulated to provide a complete diet as recommended by the Elephant SSP Nutrition Advisor.

3.1.2. There are no specific standards for elephant nutrition at this time, but see Dierenfeld (1995), Oftedahl et al. (1996) and Ullrey et al (1997).

3.2. *Medical Management*

3.2.1. A veterinarian with experience in large mammal medicine must be on call at all times to deal with routine elephant health evaluation and treatment and medical emergencies.

3.2.2. Each elephant must be given a thorough annual physical examination (Mikota et al. 1994).

3.2.3. All elephants must be visually inspected on a daily basis (EMA 1999). A general assessment must be made and any unusual activities should be recorded in the daily log at each inspection. Specifically, reports should include observations such as condition of urine and feces, eating and drinking patterns, administration of medications (if any), and general condition and behavior.

3.2.4. A veterinarian or trained veterinary technician must perform fecal examinations to look for parasites and other problems at least twice a year (Samuel et al. 2001). Results should be recorded.

3.2.5. All elephants must be trained to permit a complete body daily exam (include feet, eyes, ears, open mouth and tongue, teeth, and tusks) for any sign of abnormalities. Results should be recorded.

3.2.6. All elephants' body weight must be assessed and recorded at least twice a year (EMA 1999) through actual weighing or through the use of standardized body measurement tables, photographs, or similar, previously validated techniques (e.g., Nirmalan and Sreekumar 1990).

3.2.7. For management purposes, all elephants must be trained to accept injections, oral medications, insertion of ear or leg vein catheters, treatment of wounds, enemas, and urogenital examinations (Mikota et al. 1994, EMA 1999).

3.2.8. All elephants must be trained to accept regular collection of blood, urine, feces, saliva, semen, skin biopsy, and temporal gland secretion (Brown 1998, EMA 1999). Biological specimens should be stored according to the SSP Veterinary Advisor's guidelines on biomaterials collection.

3.2.9. All elephants' skin must be thoroughly inspected on a daily basis and cared for as needed through bathing, removal of dead skin, and treatment of dry skin or other skin problems (Mikota et al. 1994, EMA 1999).

3.2.10. Each elephant facility must have a written protocol for routine foot care and show evidence of its implementation (Miokota et al. 1994, Csuti et al. 2001). This protocol must include daily cleaning and inspection of each elephant's feet.

3.2.11. Baseline foot radiographs or thermographs of all adult elephants must be taken and kept on file. In some facilities, it may be appropriate to annually monitor selected elephants (i.e., those that have a history of chronic foot problems) (Csuti et al. 2001).

3.2.12. A written daily exercise program for each individual animal must be designed and followed (Taylor and Poole 1998). The program should be developed in consultation with the elephant manager, elephant handlers, and the staff veterinarian(s).

3.2.13. When forming new herds, Asian and African elephants should not be placed together in the same enclosure. Herpes viruses endemic to one species can be fatal in the other (Richman et al. 1996, 1999). In addition, there is concern that behavioral differences between the two species may lead to problems with dominance and aggression (Hutchins and Smith 1999).

3.2.14. Institutions must adhere to USDA APHIS requirements for testing and treatment of tuberculosis (USDA APHIS 2000, Mikota et al. 2000).

**4. Reproduction**

4.1. Each male and female elephant of reproductive age (8 to 35 years) must have an initial reproductive assessment and follow-up assessments on a regular basis by transrectal ultrasound to verify reproductive status and assess overall reproductive health (Hermes et al. 2000, Hildebrandt et al. 2000 a,b). Exceptions include elephants with known reproductive problems, actively breeding animals, or those with documented medical/behavioral conditions that preclude them from breeding.

4.2 Each male and female elephant of reproductive age (8 to 35 years) must have hormone (progesterone or testosterone) values assessed through weekly (or bi-weekly) collection of blood samples (Brown 1998, 2000). Exceptions are elephants with known reproductive problems or those with documented medical/behavioral conditions that preclude them from breeding.

**5. Behavior management**

5.1. *Training*

5.1.1. Electrical devices designed for use on livestock, such as commercially manufactured elec-

tric prods and shocking collars/belts, are prohibited as routine training tools or for handling animals during exhibition. Electric prods are permissible only as an emergency safety device; however, their use is restricted to situations in which keepers feel the imminent need to defend themselves against elephant attacks, or to protect an elephant from possible injury (see Schanberger et al. 2001).

5.1.2. Elephant training terminology and descriptions of specific behaviors are outlined in the *AZA Schools for Zoo and Aquarium Personnel Principles of Elephant Management (PEM) Course Notebook* (AZA Board of Regent's 2001). Trained behaviors should allow the elephant staff access to the animal in order to accomplish all necessary animal care and management procedures and permit inter-institutional consistency. The PEM-recommended list of commands and their corresponding behaviors are ones that every elephant and elephant keeper must know so that basic husbandry and veterinary practices can be accomplished. Behaviors should be reinforced so that all elephants attain close to 100% compliance upon request of the elephant staff (Sevenich et al. 1998).

Appropriate elephant training may employ several training aids or "tools" (see PEM Course notebook for a list and description of some elephant training tools and procedures). The goal of a good trainer is to be able to reduce the amount of time any particular training aid is used (Roocroft and Zoll 1994).

The AZA considers the following training tools/techniques to be inappropriate for use at member institutions:

a. Insertion of any implement into any bodily orifice, unless directed by a veterinarian specifically in connection with training for a medical or reproductive procedure.
b. Striking an elephant with anything more substantial than an ankus (a traditional training tool used by elephant trainers)
c. Striking an elephant with any sharp object, including the hook of an ankus (Fowler 1995).
d. Striking an elephant on or around any sensitive area, such as the eyes, mouth, ears, or genital region.
e. No tools used in training should be applied repeatedly and with such force that they cause any physical harm to an animal (i.e., breaking of the skin, bleeding, bruising, etc.).
f. Withholding or reducing an animal's daily-recommended amount of food and or water.
g. Withholding veterinary care for any reason.

If properly executed training procedures are ineffective in eliminating aggressive or inappropriate behavior in a given animal, institutions should consider other alternatives, including transfer to a facility with more experienced staff or a different management system. Protracted and repeated use of corporal discipline in training is of serious ethical concern and AZA considers abusive training practices to be unacceptable. Further, elephants that are untrained, unexercised, or unable to complete minimum behavioral requirements may be considered neglected and thereby abused.

5.2. *Management Systems*—Different elephant management systems have both advantages and disadvantages (Desmond and Laulie 1991, Doyle 1993, Preist et al. 1998, Schmid 1998). AZA standards for elephant management recognize that a diversity of approaches exist, but encourage members to continue to experiment with the goal of maximizing elephant health and reproduction and minimizing risk of injury to keeper staff (Lenhardt 1991, 2001, Chapple and Ridgway 2001). System definitions have been defined in the PEM Course and are as follows:

Free Contact—The direct handling of an elephant when the keeper and elephant share the same unrestricted space. Neither the use of chains nor the posture of the elephant alters this definition.

Protected Contact—Handling of an elephant when the keeper and the elephant do not share the same unrestricted space. Typically in this system the keeper has contact with the elephant through a protective barrier of some type while the elephant is not spatially confined and is free to leave the work area at will. This includes confined contact, where the handling of an elephant through a protective barrier where the elephant is spatially confined, as in an Elephant Restraint Device (ERD).

5.3. *Management Protocols*—Each AZA member institution and related facility that holds elephants must have a written elephant management policy. This policy must be consistent with AZA standards for elephant management and care, and must, at minimum, include a description of the institution's:

a. Elephant management program's missions and goals (EMA 1999).
b. Elephant management policies, including guidelines for handling, training, and translocation (EMA 1999).
c. Plan to separate animals from each other, safely manage elephants that are aggressive toward other elephants, safely move elephants from

one location to another, and safely manage elephants that are aggressive toward humans (EMA 1999).

d. Staff management policies, including guidelines for keeper safety (EMA 1999).
e. Individual elephant profiles and incident reports for all cases in which elephants show aggression toward keepers or the public, regardless if any injury actually resulted.
f. Emergency response protocol. Institutions should be able to demonstrate readiness to respond to an emergency situation, such as an elephant escape or keeper injury (EMA 1999).

5.4. *Safety*

5.4.1. All elephant-holding institutions must undertake at least a semi-annual elephant facility and program safety assessment, identify safety needs, and fully implement any corrective measures. Each facility shall establish a safety assessment team. The team may include elephant staff, management staff, animal health care staff, and experts in the area of risk management and safety. Each facility should establish the make-up of the team based on its own needs and resources. A written record must be kept for each inspection and that record be reviewed and its recommendations acted upon.

5.4.2. In the interest of public safety, AZA strongly discourages visitor-elephant interactions, outside of the primary enclosure. AZA strongly discourages the practice of walking elephants in public areas during public hours (BOD 3/25/03).

5.4.3. In the interest of safety, AZA strongly encourages members to discontinue public elephant rides (BOD 3/21/00).

5.5. *Restraint*

5.5.1. Chaining is acceptable as a method of temporary restraint (Fowler 1995). However, elephants must not be subjected to prolonged chaining (for the majority of a 24-hour period) unless necessary for veterinary treatment or transport. Institutions that regularly use chains for some portion of a day must alternate the chained foot on a daily basis. All new construction and major renovations must be constructed in a manner that minimizes or eliminates the need for chaining (Schmid 1995, Gruber et al. 2000).

**Note: If AZA policies on chaining require new construction, rather than procedural changes, then institutions will have five years to comply with this requirement. Plans must be in place within three years and institutions must apply for a variance from the AZA Accreditation Commission.

5.5.2.All elephant holding facilities should install an Elephant Restraint Device (ERD) (Schmidt et al. 1991). However, all bull-holding facilities, as well as those that manage elephants in protected contact, must have an ERD. Use of the ERD should not be weather dependent.

5.5.3.Each elephant must be trained to enter and stay in the ERD, if one is available, for husbandry, veterinary, reproductive assessment, and other procedures to occur in a safe and efficient manner (Schmidt 1991).

5.5.4.If a facility does not have an ERD, staff must demonstrate a method of restraint that allows necessary husbandry, veterinary, and reproductive procedures to occur in a safe and efficient manner (Fowler 1995).

## 6. Staff Organization and Training

6.1. Each institution must have one person, designated as the elephant manager. This individual is responsible for (1) staff training; (2) developing and maintaining the program; and (3) communicating with others about the elephant program. The elephant manager must also demonstrate knowledge about all emergency protocols and continually improve elephant management techniques as the industry standards evolve.

6.2. All elephant managers must attend the AZA Principles of Elephant Management Course (BOD 3/25/03), preferably within 18 months following acceptance/promotion to the position. In addition, every elephant keeper is encouraged to attend this course. The BOD directs the Board of Regents to develop a mechanism for the PEM graduates to remain current in best practices in elephant management (BOD 3/25/03).

6.3. The BOD instructs the Board of Regents to hold best practices workshops on elephant management systems and transitioning from on management system to another (BOD 3/25/03).

## 7. Conservation, Education, and Research

7.1. *Education Programs*

7.1.1. Every institution should institute a program to educate zoo visitors about elephant and elephant conservation issues (EMA 1999, Smith and Hutchins 2000). Assistance is available from the Elephant SSP Education Advisor

7.1.2. Every institution should have up-to-date educational graphics and/or information about elephants on display to the public.

7.2. *Conservation and Research Activities*

7.2.1. AZA zoos that currently exhibit or desire to exhibit elephants should make every effort to maintain elephants in their collections so that they can contribute to conservation through public education, scientific research, and the support of field conservation. Elephants are an important flagship species and the cornerstone

of many members' African and Asian exhibit areas. (BOD 3/21/00)

7.2.2. Every institution should contribute in some way to elephant research activities (Keele and Dimeo-Ediger 1997, EMA 1999, Smith and Hutchins 2000). Involvement in one or more of the following disciplines is strongly recommended: behavior, cognition, reproduction, communication, enrichment, health (disease/pathology, nutrition), and education.

7.2.3. Every institution should contribute in some way to *in situ* conservation of elephants and their habitats (EMA 1999, Smith and Hutchins 2000).

7.2.4. AZA members are strongly encouraged to provide financial, personnel, logistical, and other support for priority research and conservation initiatives listed in the AZA Elephant SSP/TAG Action Plan (Wiese and Hutchins 1994).

**8. Cooperative Management** (BOD 3/21/00)

8.1. *SSP Participation*

8.1.1. SSP participants should be given highest priority in elephant dispositions, whether through breeding or importation.

8.1.2. AZA institutions should cooperate among themselves to pursue self-sustainability with their elephant populations. Since self-sustainable elephant populations are not possible currently within AZA, then cooperation with outside organizations should be considered on a case-by-case basis.

8.1.3. AZA zoos may provide elephants or their gametes to approved non-members on a case-by-case basis.

8.2. *Importation*

8.2.1. All elephant imports must be approved within the AZA Elephant SSP/TAG. Periodic importation may be used as a way to maintain population viability in the North American Elephant SSP/TAG population. The SSP/TAG and participating institutions will employ a combination of breeding and importation with the goal of eventually creating a self-sustaining population. When acquiring elephants for the SSP/TAG, first consider captive animals in substandard conditions in North America, then captive animals outside the U.S., then wild animals surplus to the needs of the managed population or those to be captured or killed because of human-animal conflicts (especially those that are going to be killed).

8.2.2. An effort should be made to assess the potential for cooperating with sister organizations, such as the European Association of Zoos and Aquariums (EAZA).

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## Appendix

# 11

**Guidelines for Elephant Management and Care**
**The Elephant Managers Association (EMA)**
**300 Parkside Ave., Buffalo, NY 14214**
**http://www.elephant-managers.com/**

The following guidelines apply to the husbandry and management of both African (*Loxodonta africana*) and Asian (*Elephas maximus*) elephants. The intelligence, strength, and social needs of these magnificent animals can pose many challenges for managers of elephants in human care. Facilities that manage elephants should therefore understand the substantial human, financial, and ethical commitments involved in appropriately maintaining elephants. These guidelines reflect present day practices, which are based on current scientific data and the cumulative experience of our membership. These guidelines were developed to optimize the animals' physical and psychological health, environmental conditions, and to maximize the educational and scientific value of elephants in human care. These guidelines have been developed to help assist facilities that currently manage or are planning to maintain elephants, as well as regulatory agencies with jurisdiction and oversight over the care and handling of elephants in human care.

These guidelines recognize and reflect a broad range of elephant management styles; however, the Elephant Managers' Association (EMA) believes an elephant management facility that does not operate consistently within these guidelines should be prepared to fully explain and, if necessary, defend their practices. Like any living document, the EMA guidelines will be reviewed yearly under the direction of the EMA board of directors. They will be updated as the knowledge base expands to incorporate new techniques and practices.

### PERSONNEL/PROTOCOLS

1. Each institution should have one person designated as the elephant manager. This person oversees the day-to-day management of the elephants. This individual is responsible for staff training, developing and maintaining the program and communicating with others about the elephant program.
2. The elephant manager should demonstrate knowledge about all emergency protocols and continually improve elephant management techniques as the industry guidelines evolve.
3. All elephant managers are encouraged to develop their skills by visiting other facilities, attending the Elephant Managers Association annual conference, and elephant training schools such as Riddle's Elephant Management School and AZA Principles of Elephant Management Course.
4. Each facility is encouraged to develop and implement a personnel-training protocol to document new employee training and endorsement as a qualified elephant handler.
5. It is recommended that a minimum of two qualified members of the elephant staff should be present during any contact with elephants. A qualified keeper is a person the facility acknowledges as a trained, responsible individual, capable of and specifically experienced in the training and care of elephants.
6. Each facility should have a written elephant management policy. The management policy should include, but is not limited to, the following:
   - Elephant management program's missions and goals
   - Elephant management policies, including guidelines for handling and training the elephant
   - Plan to separate animals from each other, safely manage elephants that are aggressive toward other elephants, safely move elephants from one location to another, and safely manage elephants that are aggressive toward humans
   - Incident reports for all cases in which elephants show aggression toward keepers or to the public
   - Emergency response protocol (facilities should be able to demonstrate readiness to respond to an

emergency situation, such as an elephant escape or keeper injury.)
- Safety protocols for visitor-elephant interactions and elephant rides
- Behavioral profile of each individual elephant updated annually
- Environmental enrichment plan for elephants with evidence of implementation
- Protocol for routine foot care including daily cleaning and inspection of each elephant's feet and evidence of its implementation
- Daily exercise program for each individual animal

7. All elephant facilities should undertake a regular elephant facility and program safety assessment. The safety assessments should be conducted semi-annually, identify safety needs and implement any corrective measures. It is recommended that each facility establish a safety assessment team. The team may include elephant care staff, management staff, animal health care staff, and experts in the area of risk management and safety. Each facility should establish the make-up of the team based on its own needs and resources. A written record should be kept for each inspection. The appropriate staff members should review the record and its recommendations acted upon accordingly.

## TRAINING

Trained behaviors should allow the elephant care staff access to the animal in order to accomplish all necessary animal care and management procedures and permit consistency. The EMA recommended list of commands (see addendum) and their corresponding behaviors are ones that every elephant and elephant keeper should know so that basic husbandry and veterinary practices can be accomplished. Behaviors should be reinforced so that all elephants attain close to 100% compliance upon request of the elephant staff.

Training is an important component of any elephant management program, however, protracted and repeated use of corporal discipline in training is of serious ethical concern and the EMA considers abusive training practices to be unacceptable. Further, elephants that are untrained, unexercised, or unable to complete minimum behavioral requirements may be considered neglected and thereby abused. If properly executed training procedures are ineffective in eliminating aggressive or inappropriate behavior in any given elephant, facilities should consider other alternatives, including transfer of the elephant to a facility with more experienced staff or a different management system.

All elephant training management systems have both advantages and disadvantages. The EMA guidelines for elephant management recognize that a diversity of approaches exists along the continuum of elephant management. The EMA also recognizes that the best elephant management system may be a combination of systems along the continuum. The EMA encourages facilities to continue to evaluate their management practices with the goal of maximizing elephant health, welfare and reproduction and minimizing risk of injury to keeper staff or to the elephants.

The EMA considers the following training tools/techniques to be *inappropriate*:

- Insertion of any implement into any bodily orifice, unless directed by a veterinarian specifically in connection with training for a medical or reproductive procedure.
- Striking an elephant with anything other than a guide.
- Striking an elephant with any sharp object, including the hook of a guide.
- Striking an elephant on or around any sensitive area, such as the eyes, mouth, ears, or genital region.
- Applying a tool used in training repeatedly and / or with such force that physical harm to an animal results.
- Withholding or reducing an animal's daily-recommended amount of food and/or water, unless directed by a veterinarian.
- Withholding veterinary care for any reason.

## TOOLS

There are many tools that are used in the care and management of elephants. It should be noted that any tool can be misused and every keeper should be taught the proper application of each tool.

1. Management Tools
   - Guide: A 'guide,' 'ankus,' or 'elephant hook' is a traditional tool used for directing elephant's behavior. It is used on specific points on the elephant's body to cue a desired behavior.
   - Target: A tool the elephant is conditioned to touch or move towards. Targets have been made out of different materials such as a rubber disk or a pole and a ball on the end. Application of this tool is not limited to a man made tool. An example would be the use of a finger being the target to move the elephant's tongue aside for a mouth exam.
   - Bridge (whistle/clicker/voice): A cue tells the elephant that they have achieved the desired behavior.
   - Ropes, Block & Tackle: These may be used to direct or aid an elephant when training a new behavior. They also can be used to assist or raise debilitated or partially anesthetized animals to their feet to help ensure their recovery.

- Tub, Balance Beam, Spindle: Tools that have been used for daily exercise programs and educational demonstrations. Some of these have also been used to enable foot care on elephants.

2. Restraint
   a. Chaining is an acceptable method of temporary restraint. However, facilities should limit the time elephants spend tethered unless necessary for veterinary treatment or transport. Leg chains are used to limit animal's movements, for example, during husbandry and medical procedures and to separate animals. Leg chains should be alternated from left front/right rear to the opposite front and rear legs every other day. The chains should be long enough to allow the elephants to easily lie down. All chains and attachment devises should be inspected daily, and staff should be trained in their proper use.
   b. An Elephant Restraint Device (ERD) is a device used to keep an elephant in a designated space in order to facilitate husbandry and medical procedures. There are many different designs of ERDs: stationary, electric, hydraulic and even ones capable of turning an elephant on its side. All elephant facilities should install an Elephant Restraint Device (ERD). However, it is strongly recommended that every facility managing bulls or elephants in protected contact have an ERD. Use of the ERD should not be weather dependent. Each elephant should be trained to regularly enter and stay in the ERD to allow for basic husbandry needs, veterinary procedures, reproductive assessments, and other procedures to occur in a safe and efficient manner. If a facility does not have an ERD, staff should demonstrate a method of restraint that allows necessary husbandry, veterinary, and reproductive procedures to occur in a safe and efficient manner.

2. Safety Tools:
   a. Pepper Spray
   b. Fire Extinguisher
   c. Electrical devices designed for use on livestock, such as commercially manufactured electric prods, may be used to fend off an elephant attack
   d. Tranquilizer gun to tranquilize an escaped or highly agitated elephant
   e. Elephant gun to dispatch an escaped elephant when there is immediate danger to human life

## HUSBANDRY

- All elephants should be visually inspected on a daily basis. A general assessment should be made and any unusual activities should be promptly recorded in the daily log. Specifically, reports should include observations of the individual elephants such as condition of urine and feces, eating and drinking patterns, administration of medications (if any), and general condition and behavior.
- All elephants' skin should be thoroughly inspected on a daily basis and cared for as needed through bathing, removal of dead skin, and treatment of dry skin or other skin problems.
- All elephants should be trained to permit a complete daily body exam (including feet, eyes, ears, open mouth and tongue, teeth, and tusks) for any sign of abnormalities. Results should be documented in the elephants' health records.

## FACILITIES

- If the captive elephant population is to become sustainable, it is necessary to create housing for many more adult males. All facilities should consider including holding space for adult males. Holding space for males must be designed to best care for the male elephant in musth, allowing him space to move and exercise safely.
- Facilities should have the ability to manage social compatibility as well as dominance and aggression within an elephant group.
- Facilities should have the ability to manage introductions and separations such as a new female to a herd, females to males for breeding, a newborn calf to its mother, and a calf and mother to the herd.
- Facilities should provide an opportunity for each elephant to exercise and to interact socially with other elephants.
- All facilities should have the ability to separate and isolate animals to address behavioral concerns or allow veterinary procedures to occur.
- All enclosures should be cleaned and disinfected daily. Frequent manure removal during the day is recommended and may be necessary for both sanitary and aesthetic conditions.

### Indoor

- Indoor space should provide adequate room for elephants to move about and lie down without restriction.
- Mature elephants can reach items with their trunks at a vertical height of 20 ft (6.1 m) and potentially higher. Consideration of this should be given with regard to ceiling heights and fixtures (e.g., lights, heating units, plumbing, etc.) so that elephants do not harm themselves or the facility.
- Indoor holding areas should be ventilated and heated to a minimum temperature of at least 55 degrees F (12.8 degrees C) at all times of the year.
- Natural daylight cycles are adequate for elephants. Indoor areas should be well illuminated during daylight hours, followed by a period of darkness. The use of natural light utilizing skylights and/or windows is suggested.
- Standing water in indoor floor areas can cause foot problems and become a breeding ground for contam-

inants. Floors should therefore be impervious to water, quick to dry, and sloped to a drain. Floor surfaces should be relatively smooth, but not smooth enough so that they become slippery when wet. Conversely, very rough surfaces may cause excessive wear or irritate footpads.

### Outdoor

- Outdoor areas should have enough space for animals to get away from each other if they wish and be large enough for adequate exercise opportunities.
- Environmental enrichment programs and the physical condition of the elephants should also be taken into consideration when evaluating space.
- During daylight hours, healthy elephants kept outdoors can tolerate a wide range of temperature extremes. Provisions should be made to protect elephants from adverse weather, including cold winds, chilling rain, sleet, sun, heat, etc. Elephants kept outdoors without access to heated facilities should be monitored frequently at temperatures below freezing.
- While outdoors, all elephants should have access to shade during daylight hours in temperatures above 80 degrees F (27 degrees C). Elephants kept outdoors should be monitored frequently at temperatures above 90 degrees (32 degrees C).
- Outdoor yard surfaces should consist primarily of natural substrates (e.g., soil, sand, grass) that provide good drainage and have a cleanable, dry area for feeding.
- Elephants should be kept outdoors on natural substrates as much as possible.

Facilities should consider designing elephant areas that allow elephants outdoor access twenty-four hours a day—weather, health, and safety issues permitting.

### Barriers

- Elephant containment barriers should be in good condition and able to prevent elephant escapes. A wide variety of building materials can be used as long as they are able to withstand the elephant's strength, contain the elephant in a specific space, and allow adequate space between the elephant and the public.
- Door and gate design is extremely important to ensure the safety of both elephants and keeper staff. Doors and gates should be engineered to withstand extreme force. If mechanical opening devices, such as hydraulic or electrically powered drives are used, they should be able to be operated manually or with a backup generator in the case of a power failure.
- Doors should be designed so that the person operating the doors is able to see the doorway, either directly or via indirect means such as closed circuit cameras.
- All doors should be designed so that they can be stopped immediately in the event an elephant steps in the path of an operating door.
- The use of poorly designed dry moats (moats that are steep, deep, narrow-sided, and hard-bottomed) as primary containment can be particularly dangerous for elephants and their use should be carefully considered. A written elephant extraction protocol should be in place in any facility using moats directly around elephant areas.
- Electric fences and similar devices have been used successfully to protect trees and like items from elephants. If used as a primary containment barrier, elephants should be monitored constantly.

### Behavior

- Elephant management facilities should make every effort to maintain elephants in social groupings. It is inappropriate to keep highly social female elephants singly. Elephant care staff should be aware of each animal's social compatibility and the dominance hierarchies of the herd.
- The minimum age offspring should remain with their mothers is two years. Some flexibility is necessary in cases of maternal rejection and when infants cannot be reestablished in their social group.
- Adult males may be housed alone, but not in complete isolation. Opportunities for tactile, olfactory, visual, and/or auditory interaction with other elephants should be provided.

## REPRODUCTION

- Each male and female elephant of reproductive age (approximately 8 to 35 years) should have hormone (progesterone or testosterone) values assessed through weekly (or bi-weekly) collection of blood, urine or fecal samples. Exceptions are elephants with known reproductive problems or those with documented medical/behavioral conditions that preclude them from breeding.
- Each male and female elephant of reproductive age (approximately 8 to 35 years) should have an initial reproductive assessment and follow-up assessments every 2–3 years by transrectal ultrasound to verify reproductive status and assess overall reproductive health. Exceptions include elephants with known reproductive problems, actively breeding animals, or those with documented medical/behavioral conditions that preclude them from breeding.

## VETERINARY CARE

- A veterinarian with experience in large mammal medicine should be on call at all times to perform elephant health evaluations, oversee treatment and medical emergencies.
- A veterinarian should give each elephant a thorough annual physical examination.

- A veterinarian or trained veterinary technician should perform fecal examinations to look for parasites and other potential problems at least twice a year. Results should be documented in the elephants' health records.
- For management purposes, all elephants should be trained to accept injections, oral medications, insertion of ear or leg vein catheters, treatment of wounds, biological sample collection, enemas, and urogenital examinations. All elephants should have serum samples obtained at least quarterly and stored for future reference.
- It is recommended that baseline foot radiographs or thermographs of all adult elephants be taken and kept on file. In some facilities, it may be appropriate to annually monitor selected elephants (i.e., those who have a history of chronic foot problems).
- Facilities must adhere to USDA APHIS requirements for the annual testing and for treatment of tuberculosis.

## NUTRITION

- High quality and nutritionally correct food should be provided daily in sufficient quantities to maintain elephant health and appropriate weight and should be formulated to provide a complete elephant diet.
- Elephants should have daily access to clean, fresh drinking water. When water containers are used, drinking water containers should be cleaned and refreshed daily.
- Fresh browse and produce should be used often as dietary supplements and enrichment for the animals.
- All elephants' body weight should be assessed and recorded a minimum of twice a year through actual weighing, or through the use of standardized body measurement tables, photographs, or similar, previously validated techniques.
- Obesity is a major concern for the long-term health of the captive elephant population.

## CONSERVATION, EDUCATION, AND RESEARCH

The goal of Education, Conservation and Research Activities is to enhance the appreciation and understanding of elephants and their ecosystems and support elephants and habitat in range countries. An informed visitor is more likely to support research and conservation of elephants and of their habitat.

- Every elephant facility should institute a program to educate visitors and promote an improved understanding about elephants and elephant conservation issues.
- Every elephant facility should have up-to-date educational graphics and/or information about elephants on display to the public.
- Facilities that currently manage or desire to manage elephants should make every effort to contribute to conservation through public education, scientific research, and the financial support of field conservation projects.
- It is the responsibility of every elephant facility to contribute in some manner to in situ and ex situ research and conservation of elephants.

## Appendix

# 12

**Guidelines for Comprehensive Elephant Health Monitoring Program Elephant Species Survival Plan, American Zoo and Aquarium Association May 2005**

Routine health monitoring should be performed on all elephants on an ongoing basis. Animals should be trained to permit sampling and examination. The following protocol advises that specific baseline laboratory tests be performed for the purpose of evaluating current health status. Additional tests are recommended to increase baseline information on other diseases to determine their significance to elephant health. The final decision for specific procedures should be made by the institutional animal care and veterinary staff based on individual circumstances. For additional information, refer to the *Elephant Husbandry Manual,* AZA Standards for Elephant Management and Care [Appendix 10, this book], and the AAZV Preventive Medicine Recommendations. Additionally, it is recommended that the veterinarian review the behavioral profile of the individual animals with elephant management staff on a regular basis.

Minimum Database:

1. Signalment—age, sex, origin, studbook #, ISIS #; picture of individual (as viewed from the front and sides) should be included in the permanent record.
2. Anamnesis—summary of information regarding health screens, medical problems, diagnostic test results, and treatment over the previous year (complete "Individual Annual Elephant Medical Survey" form and send to SSP Veterinary Advisor—will be available in 2006).
3. Complete physical exam by a veterinarian familiar with elephant health problems. This should include a review of all systems.
4. Body weight—actual weight should be recorded at least annually.
5. Blood collection
   a. Complete blood count (CBC), serum chemistry panel, fibrinogen, serum protein electrophoresis
   b. Serologic (ELISA) test for elephant herpesvirus—contact Erin Latimer to submit samples, or Dr. Laura Richman (1).
   c. Bank a minimum of 10–20 ml serum (duplicate sample for SSP serum bank)—all banked samples should be labeled with species, studbook #, age, sex, and date collected. Use submission form for serum samples sent to the SSP serum bank (maintained at Disney's Animal Kingdom).
6. Serum progesterone analysis in females—Serial samples should be collected weekly on an ongoing basis to evaluate estrous cycles (2). Assays can be performed at the National Zoo's Conservation & Research Center or Southwest Missouri State University. Contact specific institutions (Dr. Janine Brown—CRC (National Zoo), Dr. Dennis Schmitt—S.M.S.U.) for submission instructions (see protocol below).
7. Fecal analyses
   a. Parasite screen—fecal samples should be collected at least semiannually; direct, flotation, and sedimentation should be performed on every sample to detect intestinal parasitism.
   b. Enteric pathogen screen—aerobic culture of feces for enteric pathogens should include special media for the detection of *Salmonella* spp. Because Salmonella organisms may be shed intermittently, at least 3–5 fecal cultures should be performed (may be done on consecutive days) on an annual basis.
8. TB culture—refer to the current USDA Guidelines for the Control of Tuberculosis in Elephants (3). Protocol can be accessed on the USDA website: www.aphis.usda.gov/ac/ElephTBGuidelines2003.html. At this time, annual trunk wash cultures are the only required test; however, collection of other samples for research is strongly encouraged (minimum 2 ml serum collected at the time of trunk washes should be

sent to Dr. Michele Miller for use in validating other serologic tests for TB diagnosis).

a. Samples for cultures must be collected under the direct supervision of a licensed veterinarian.
b. Three trunk wash samples should be collected on separate days, ideally within a 7-day period. Trunk swabs are no longer acceptable.
c. All samples should be frozen immediately after collection and shipped frozen.
d. Ship by overnight express to NVSL (or other laboratory facility offering comparable procedures). Request mycobacterial culture with speciation (use VS Form 10-4 submission form for NVSL).

9. Vaccinations
   a. Tetanus toxoid*—annual vaccination with a commercial equine product is recommended. Follow label instructions for product use (usually 1 ml IM). Data are insufficient at this time to determine adequate protective vaccine doses and titers.
   b. Rabies vaccine*—vaccination with a commercial killed rabies product approved for horses should be considered if the animal resides or will be traveling to an endemic area. Follow label instructions for product use (usually 2 ml IM). Vaccination with Imrab 3 has induced detectable titers to rabies virus in African elephants (M. Miller, pers. comm.).** Biannual vaccination is recommended. Data are insufficient at this time to determine adequate protective vaccine doses and titers.

*Both tetanus and rabies have been reported to occur in elephants (4,5).

**This is based on the persistence of titers (>1:50) in one herd of African elephants in response to Imrab 3 for more than 2 years.

Additional Preventive Health Recommendations:

1. Serological screening for EMC (encephalomyocarditis virus), leptospirosis (multiple serovars), and WNV (West Nile Virus). Although these tests are not species-specific and have not been validated for elephants, they may detect cross-reactive antibodies in exposed animals. The presence of antibodies does not necessarily denote infection/disease. Encephalomyocarditis virus may cause clinical disease and death in elephants (6). Antibodies to leptospirosis have been detected in both Asian and African elephants (7, 8). At the time of this writing, EMC serologic testing was not available. Insufficient data is available at this time to determine the significant of WNV antibodies in elephants; it is important to include the history of exposure and vaccination to WNV when interpreting results.
2. Serum vitamin E levels—submit heparinized plasma to Michigan State University.
3. Reproductive tract examination—whenever feasible, a complete reproductive examination should be conducted which includes transrectal ultrasound, semen collection and analysis, serum collection (weekly best, or bi-weekly to monthly) for testosterone analysis, cytology and microbial cultures of the lower urogenital tract (to be screened for bacteria, Chlamydia, protozoa, and Herpesvirus). Herpesvirus has been identified in biopsies of vaginal lymphoid patches in an African elephant (9). A high prevalence of uterine leiomyomas has been observed in captive Asian elephants and could be detected via transrectal ultrasound (10). Since both of these conditions have potentially significant effects on reproduction, a careful evaluation is warranted if the animal is being considered for breeding. All elephants (male and female) over the age of 5 years should have both ultrasound and hormonal (testosterone in males; progestins/LH in females) assessments performed.
4. Urinalysis—fluid and sediment evaluation of clean voided sample; +/− microbial culture.
5. Foot radiographs—baseline radiographs of all feet are strongly recommended; see Gage for description of technique (11). It may be appropriate to annually monitor selected elephants (i.e., those that have a history of chronic foot problems).
6. Ancillary diagnostic tests for tuberculosis—ELISA, etc. recommended for data gathering; see Guidelines for the Control of Tuberculosis in Elephants for current recommendations (3).
7. Other vaccination regimens will depend on regional requirements and exposure risks (consider vaccination for equine encephalitis viruses, Clostridial diseases, Leptospirosis). Insufficient information is available at this time to provide a recommendation for West Nile Virus vaccination of elephants. Contact the SSP veterinary advisor for current information.

**Elephant Serum Bank Submission Form**
**American Zoo and Aquarium Association**
**Elephant Species Survival Plan**

Institution/owner: _____
Submitter: _____
Address: _____
_____
Tel:_____Fax:_____Email: _____

Animal Information

Asian [ ] African [ ] ISIS # _____ Studbook # _____
Name _____ Age:_____ [ ] actual [ ] estimate
Sex: [ ] male [ ] female

SAMPLE COLLECTION INFORMATION

Date of sample collection: _____ Time of collection: _____
Site of sample collection:[ ] ear vein [ ] leg vein [ ] other: _____
Health status of animal: [ ] normal [ ] abnormal
Fasted: [ ] no [ ] yes—how long _____
Weight _____ [ ] actual [ ] estimated
Type of restraint: [ ] manual [ ] anesthetized/sedated [ ] behavioral control
Temperament of animal: [ ] calm [ ] active [ ] excited

Type of blood collection tube:
[ ] no anticoagulant (red-top)
[ ] EDTA (purple)
[ ] heparin (green)
[ ] other: _____
Sample handling: [ ] separation of plasma/serum by centrifugation
(check all that apply) [ ] stored as whole blood
[ ] frozen plasma/serum
[ ] other—describe _____

**TB EXPOSURE STATUS**

[ ] Known infected animal
[ ] Known exposure to a culture positive source within the past 12 months
[ ] Known exposure to a culture positive source within the past 1–5 years
[ ] No known exposure to a culture positive source in the last 5 years

**TREATMENT INFORMATION**

Is elephant currently receiving any medication or under treatment? [ ] yes [ ] no
If yes, please list drugs and doses: _____
_____
_____
_____

Time between blood collection and last treatment: _____

---

Ship samples overnight frozen with shipping box marked “PLACE IN FREEZER UPON ARRIVAL”

**Send completed form with samples to:**

Dr. Michele Miller
Disney’s Animal Kingdom—Dept. of Vet. Services
P.O. Box 10,000
Lake Buena Vista, FL 32830-1000
(407) 939-7316; email: Michele.Miller@disney.com

TB ELISA SAMPLE SUBMISSION FORM FOR ELEPHANT SAMPLES (LAB 1)

PAGE___
OF ___

Please send samples to:
**Mycobacterium bovis Testing Laboratory**
Animal Population Health Institute
Environmental Health Bldg. RM 107
Colorado State University
Fort Collins, CO 80523-1676

Attn: Joni
Phone: (970) 491-2379; Fax: (970) 491-2940

For Questions Regarding Sample Submission, Test Results, or Interpretation Please Contact:

Dr. Scott Larsen
School of Veterinary Medicine
University of California
Davis, CA 95616
Phone: (530) 752-6059; Fax (530) 752-0414
slarsen@ucdavis.edu

LAB USE ONLY

JTPID:

Date Rec'd:

Location:

SUBMITTED BY: PHONE:

NUMBER OF SAMPLES:

CLINIC/INSTITUTION: FAX:

ADDRESS: E-MAIL:

CITY: STATE: ZIP: OWNER OF ANIMALS:

| Date Serum Taken | Elephant Name | Stud-book or ISIS # | Species | Sex | Age | Birth Date | Time at Current Facility | Trunk Wash Date | Trunk Wash Results | Skin Test Date | Skin Test Results | PCR Date | PCR Results |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
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SAMPLE SUBMISSION
1. Samples should be submitted in 12 mm × 75 mm serology tubes or 1.7 ml microcentrifuge tubes.
2. Collect blood into red-top clotting tube. Allow to clot, centrifuge, and transfer serum into another vial.
3. If possible, ship at least 3 ml of serum for each sample submitted.
4. Shipping boxes must say "REFRIGERATE UPON ARRIVAL."
5. Send via overnight shipment at 4°C or frozen. Please take care that samples will not arrive on a weekend or holiday.

## Instructions for Elephant Endotheliotropic Herpesvirus (EEHV) Sample Submission

Please submit all samples on plenty of dry ice in a sealed styrofoam container.

- For a sick elephant with suspected active EEHV infection, please send at least 1–2 ml of whole blood (EDTA, lavender-top tube). It is best to transfer the blood to a freezer-safe tube (not glass) after thoroughly mixing with EDTA. We have received broken glass tubes in the past.

- We can test any tissue for EEHV from deceased elephants, but it must be frozen (preferably in liquid nitrogen or dry ice, or store in a −70°C freezer). Heart, liver and spleen are the best organs to test for EEHV.

- We would like to test placenta from all newborn/stillborn/aborted elephants. Please freeze a 1-inch piece (preferably in liquid nitrogen or dry ice, or store in a −70°C freezer).

- For EEHV titers, please send at least 2 mls of serum (preferably more). Freeze immediately and send on dry ice.

- Please include all pertinent elephant information (including history) on the attached form.

- The best day to ship (FedEx) would be Wednesday for a Thursday arrival. Please call Erin or Laura before shipping samples:

Erin Latimer: 202-633-4252 (W) (H)
LatimerE@nzp.si.edu

OR

Laura Richman: 301-398-4741 (W)
301-253-8723 (H)
RichmanL@MedImmune.com

**FedEx to:**

**Erin Latimer/Laura Richman**
**Smithsonian National Zoological Park**
**Department of Pathology**
**3001 Connecticut Ave. NW**
**Washington, DC 20008**
**(202) 633-4252**

# Request for EEHV testing

**Department of Pathology**
**Smithsonian National Zoo**
**3001 Connecticut Ave. NW**
**Washington, DC 20008**
**(202) 633-4252**

Date _____

Requestor's name _____

Institution _____

Address _____

_____

_____

Phone/email _____

Animal Name/ISIS # _____

Age _____

Species _____

Reason for request (i.e., current symptoms of possible EEHV, or possible exposure to EEHV) _____

_____

_____

Type of samples being sent _____

Before sending samples, please call Erin Latimer 202-633-4252 (W)
703-471-2168(H)

or Laura Richman 301-398-4741 (W)
301-253-8723 (H)

Please send samples **overnight on dry ice in a sealed cooler**. The best day to send samples is Wednesday, for Thursday delivery.

## SERUM COLLECTION PROTOCOL FOR MONITORING REPRODUCTIVE ACTIVITY IN ELEPHANTS

**Janine L. Brown, PhD**
Elephant SSP Reproductive Advisor
Endocrine Research Laboratory

### Females

- Blood samples should be collected once weekly to establish if females exhibit normal ovarian cycles (e.g., estrous cyclicity). **Note:** the Elephant SSP requires that females between 8 and 35 years of age be hormonally assessed, and recommends that all elephants be monitored throughout their lifespan.

### Males

- Blood samples should be collected weekly (bi-weekly or monthly if weekly is not possible) to assess testicular steroidogenic activity. **Note:** the Elephant SSP recommends that elephant bulls be monitored throughout their adult lifespan.

### Sample collection and shipment

- For best results, allow blood to clot for ~1 hour at room temperature, or for ~2 hours at refrigerator temperature. Avoid exposing blood to ambient temperatures for longer than 3 hours (blood cells can metabolize progesterone and affect results). Plasma (collected in either EDTA or heparinized tubes) can also be analyzed, although serum is preferred.
- Centrifuge blood (~1000 × g for 10–15 min) and decant serum into a polypropylene vial (best sizes, 12 × 75 mm or 12 × 55 mm) with a tight-fitting cap that pushes or screws on and is flush with the tube (i.e., cap doesn't hang over the side of the tube). We recommend tubes with a frosted writing space and caps from the Sarstedt company (1-800-257-5101). The catalogue number for the tubes is 62.526.003 PP, and for the push caps is 65.809. Smaller tubes are okay, but do not use ones that exceed a 5-ml capacity. Information on the tube should include: animal name, date (mo/day/year), and facility name or abbreviation. Please provide a minimum 1 ml of serum for each sample. Store frozen (−20°C or colder).
- Ship samples in a styrofoam container with dry ice or cold packs. We will return box and any shipping materials. Use an overnight express courier (e.g., Airborne Express or FedEx) and ship only on Mon, Tues or Wed. Never ship on Fri or before a government holiday.
- Include in the shipment a written request as to what hormone analyses are required. If you need results immediately (emergencies only, please), let us know in the paperwork you provide. If you will be requesting Prolactin or LH (or any other protein hormone), call a week in advance so that we can schedule an iodination.
- Address samples to: Nicole Abbondanza, Conservation and Research Center, 1500 Remount Rd., Front Royal, VA 22630. Please notify us when samples are shipped (540-635-6521, ext. 225; abbondanzan@crc.si.edu).
- We will issue an invoice at the time data are sent. Please make checks payable to: Conservation & Research Center Foundation, c/o Janine Brown.
- For our records, please provide the studbook number, name and age of your elephant(s). If you have any questions, please contact Dr. Janine Brown: phone (540) 635-6586, fax (540) 635-6506, email jbrown@crc.si.edu. Lab hours are 8:00 am–5:00 pm est.

## REFERENCES

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2. Brown, J.L. 2000. Reproductive endocrine monitoring of elephants: An essential tool for assisting captive management. Special Issue on Elephant Biology. Zoo Biol 19:347–368.
3. Guidelines for the Control of Tuberculosis in Elephants. Available on the Internet: www.aphis.usda.gov/ac/ElephTBGuidelines2003.html
   For regulatory questions, contact:
   Dr. Denise Sofranko
   USDA, APHIS, Animal Care
   1629 Blue Spruce Drive, Suite 204
   Ft. Collins, CO 80524-2013
   Voice Mail: 703- 812-6682; FAX: 505-293-7466
   Email: Denise.M.Sofranko@aphis.usda.gov
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## CONTACT LIST

- Elephant SSP Veterinary Advisor
  Dr. Michele Miller
  Disney's Animal Kingdom
  Dept. of Veterinary Services
  P.O. Box 10,000
  Lake Buena Vista, FL 32830-1000
  Email: Michele.Miller@disney.com

- Elephant SSP Pathology Advisor
  Dr. Scott Terrell
  Disney's Animal Kingdom
  Dept. of Veterinary Services
  P.O. Box 10,000
  Lake Buena Vista, FL 32830-1000
  Email: Scott.Terrell@disney.com

- Elephant SSP Reproductive Advisor
  Dr. Janine Brown
  Conservation and Research Center
  1500 Remount Rd.
  Front Royal, VA 22630
  Email: jbrown@crc.si.edu

- Elephant SSP Nutritional Advisor
  Dr. Ellen Dierenfeld
  St. Louis Zoological Park
  Forest Park
  1 Government Dr.
  St. Louis, MO 63110-1396
  Email: dierenfeld@stlzoo.org

Dr. Laura Richman
RichmanL@MedImmune.com

Erin Latimer (EEHV tech)
Dept. of Pathology
Smithsonian National Zoo
3001 Connecticut Ave., NW
Washington, D.C. 20008

Dr. Dennis Schmitt
SMSU—Agriculture Dept.
217 Karl's Hall
901 South National Ave.
Springfield, MO 65804
Email: dennisschmitt@smsu.edu

National Veterinary Services Laboratories (NVSL)
Dr. Janet Payeur
1800 Dayton Rd.
Ames, IA 50011
Email: Janet.B.Payeur@usda.gov

TB ELISA—Dr. Scott Larsen
School of Veterinary Medicine
University of California
Davis, CA 95616
Email: slarsen@ucdavis.edu
See attached submission form for samples

For Leptospiral and WNV titers, contact:
Cornell University, College of Vet. Med.
Diagnostic Lab
Upper Tower Rd.
Ithaca, NY 14853

For vitamin E analysis, contact:
Michigan State University
Animal Health Diagnostic Lab
4125 Beaumont Rd.
East Lansing, MI 48910

### Additional Contacts for General Elephant Medical Information:

Dr. Susan Mikota
Director of Veterinary Programs and Research
Elephant Care International
438 N. Central Ave.
Waveland, MS 39576
Email: smikota@elephantcare.org

Dr. Genny Dumonceaux
Busch Gardens Tampa Bay
P.O. Box 9158
Tampa, FL 33674-9158
Email: Genevieve.dumonceaux@anheuser-busch.com

# Appendix 13

## QUARANTINE GUIDELINES FOR ELEPHANTS

### American Zoo and Aquarium Association Elephant Species Survival Plan

### February 2003

Due to the size, strength, and social nature of elephants, it may be logistically difficult to maintain isolation from other animals during arrival and quarantine. The Recommended Preshipment Protocol for Elephants lists a comprehensive battery of tests to detect disease prior to shipment. Since most zoological institutions will not have facilities available to safely house and manage a newly arriving elephant, it is important that the receiving institution work closely with the sending institution to ensure that all (or as many as possible) of the listed tests are conducted and results reviewed. Following the preshipment protocol may help compensate for some of the quarantine compromises that may be required. Regardless of preshipment test results, every attempt should be made to maintain some degree of physical separation from the resident elephants after arrival.

Current quarantine practices recommend a minimum 30–90 day quarantine period for most species found in zoos and aquaria. Social concerns, physical facility design, and availability of trained elephant staff may dictate a modified quarantine protocol. The final decision for specific quarantine protocols at each institution should be made by the veterinary staff in consultation with the elephant management staff. For additional information, refer to the Elephant Husbandry Manual, AZA Quarantine Guidelines, and the AAZV Preventive Medicine Recommendations.

The following guidelines provide recommendations for minimum standards for elephant quarantine.

- Whenever possible, the newly arrived elephant should be maintained with physical separation from all other resident elephants. This should include provisions to prevent contact with feed, bedding, or feces/urine between animals. One option to allow social interaction is to provide a "companion" and treat both animals as "quarantined."
- Initial visual assessment of the elephant, along with review of the medical records, to determine health status should be used to develop an individual quarantine plan.
  - Ideally, the recommended length of quarantine is a minimum of 30 days. However, this may be changed in light of social concerns or detection of abnormal health status.
  - Risk of disease transmission between animals should be balanced with the concern for well-being (physical, psychological, and social) of the elephant.
- Quarantine procedures should be planned as soon as the elephant can be safely managed and appears to be settling in the facility.
  - Thorough physical examination including a review of all systems.
  - Blood collection for CBC, serum chemistry panel, fibrinogen, serum protein electrophoresis, and serum bank.
  - Fecal collection for parasite screening should be done weekly for the first 3 weeks.
  - Fecal cultures for Salmonella should be conducted at least weekly for the first 3 weeks.
  - Any procedures that were not completed prior to transport or may have come due, such as vaccination, serologic screening, or TB testing (see "Recommended Elephant Preshipment Guidelines" [Appendix 14]).
- Release from quarantine should be the decision of the veterinary staff (after completion and review of results from any quarantine procedures), in conjunction with the assessment of the elephant management staff.

It should be emphasized that the quarantine test requirements should be conducted regardless of the preshipment testing. The stress of transport and quarantine may result in changes (for example, Salmonella shedding) that were inapparent during testing at the sending institution.

## Appendix

# 14

**RECOMMENDED ELEPHANT PRESHIPMENT GUIDELINES**
**American Zoo and Aquarium Association Elephant Species Survival Plan**

**February 2003**

Routine health monitoring should be performed on all elephants on an annual basis (see "Guidelines for Comprehensive Elephant Health Monitoring Program" [Appendix 12]). Animals should be trained to permit sampling and examination. Whenever possible, preshipment testing should be performed within 30–90 days of the anticipated shipping date (note: mycobacterial cultures require 60 days for final results). The following protocol advises that specific baseline laboratory tests be performed for the purpose of evaluating current health status. Additional tests are recommended to increase baseline information to determine their significance to elephant health. The final decision for specific procedures should be made in partnership between the shipping and receiving institutions. Any abnormal findings should be communicated to the receiving institution in a timely manner. For additional information, refer to the Elephant Husbandry Manual, AZA Standards for Elephant Management and Care, and the AAZV Preventive Medicine Recommendations.

Minimum Database:

1. Signalment—age, sex, origin, studbook #, ISIS #; picture of individual (as viewed from the front and sides) should be included in the permanent record.

2. Anamnesis—summary of information regarding previous health screens, medical problems, diagnostic test results, and treatment (complete "Individual Annual Elephant Medical Survey" form and send to SSP Veterinary Advisor—will be available in 2003). A hard copy and disc of the complete medical record should be sent to the receiving institution prior to shipment. In addition, the veterinarian should review the behavioral profile of the individual to be shipped.
   Specific areas to be included:
   a. foot/skin conditions
   b. dental/tusk conditions
   c. history of colic, diarrhea, GI parasitism (including fecal parasite screens and Salmonella cultures)
   d. serologic status, if known (EMC, elephant herpesvirus, Leptospirosis)
   e. vitamin E status, if known
   f. TB culture (dates and results)
   g. reproductive history
   h. musth history
   i. sedation/immobilization data

3. Complete physical exam by a veterinarian familiar with elephant health problems. This should include a review of all systems.

4. Body weight—actual or estimated using body measurements (1).

5. Blood collection
   a. Complete blood count (CBC), serum chemistry panel.
   b. Serologic (ELISA) test for elephant herpesvirus—contact Drs. Laura Richman or Richard Montali. See Recommendations for EEHV Testing and Transport of Elephants.
   c. Bank minimum of 10–20 ml serum (duplicate sample for SSP serum bank)—all banked samples should be labeled with species, studbook #, age, sex, and date collected. Use submission form for serum samples sent to SSP serum bank.

6. Fecal analyses
   a. Parasite screen—Fecal samples should be collected every 7 days for a total of 3 weeks; direct, flotation,

and sedimentation should be performed on every sample to detect intestinal parasitism.
   b. Enteric pathogen screen—Aerobic culture of feces for enteric pathogens should include special media for the detection of *Salmonella* spp. Since Salmonella organisms may be shed intermittently, at least 3–5 fecal cultures should be performed (may be done on consecutive days).
   c. Contact receiving institution with any abnormal results and treatments.

7. TB culture—refer to the current USDA Guidelines for the Control of Tuberculosis in Elephants (2). Protocol can be accessed on the USDA website www.aphis.usda.gov/ac/ElephTBGuidelines2003.html. At this time, annual trunk wash cultures are the only required test; however, collection of other samples for research is strongly encouraged.
   a. Samples for cultures must be collected under the direct supervision of a licensed veterinarian.
   b. Three trunk wash samples should be collected on separate days, ideally within a 7 day period. Trunk swabs are no longer acceptable.
   c. All samples should be frozen immediately after collection and shipped frozen.
   d. Ship by overnight express to NVSL (or other laboratory facility offering comparable procedures). Request mycobacterial culture with speciation (use VS Form 10-4 submission form for NVSL).

8. Vaccinations
   a. Tetanus toxoid*—current vaccination (within 12 months) with a commercial equine product is recommended. Follow label instructions for product use (usually 1 ml IM). Data are insufficient at this time to determine adequate protective vaccine doses and titers.
   b. Rabies vaccine*—current vaccination (within 12 months) with a commercial killed rabies product approved for horses should be considered if the animal resides or will be traveling to an endemic area. Follow label instructions for product use (usually 2 ml IM). Data are insufficient at this time to determine adequate protective vaccine doses and titers.

*Both tetanus and rabies have been reported to occur in elephants (4,5).

Additional Preventive Health Recommendations:

1. Serological screening for EMC (encephalomyocarditis virus), leptospirosis (multiple serovars), and WNV (West Nile Virus). Although these tests are not species-specific and have not been validated for elephants, they may detect cross-reactive antibodies in exposed animals. The presence of antibodies does not necessarily denote infection/disease. Encephalomyocarditis virus may cause clinical disease and death in elephants (5). Antibodies to leptospirosis have been detected in both Asian and African elephants (6, 7). At the time of this writing, EMC serologic testing was not available. Insufficient data is available at this time to determine the significant of WNV antibodies in elephants; it is important to include the history of exposure and vaccination to WNV when interpreting results.

2. PCR test for elephant herpesvirus—contact Drs. Laura Richman or Richard Montali (8).

3. Serum vitamin E levels—submit heparinized plasma to Dr. Ellen Dierenfeld.

4. Reproductive tract examination—a complete reproductive examination should be conducted to include transrectal ultrasound, semen collection and analysis, cytology and microbial cultures of the lower urogenital tract (to be screened for bacteria, Chlamydia, protozoa, and Herpesvirus). Herpesvirus has been identified in biopsies of vaginal lymphoid patches in an African elephant (9). A high prevalence of uterine leiomyomas has been observed in captive Asian elephants and could be detected via transrectal ultrasound (10). Since both of these conditions have potentially significant effects on reproduction, a careful evaluation is warranted if the animal is being considered for breeding. All elephants (male and female) over the age of 5 years should have both ultrasound and hormonal assessments performed (testosterone in males; progestins/LH in females). See "Guidelines for Comprehensive Elephant Health Monitoring Program."

5. Urinalysis—fluid and sediment evaluation of clean voided sample; +/- microbial culture.

6. Foot radiographs – baseline radiographs of all feet are strongly recommended (send copies of radiographs to receiving institution); see Gage for description of technique (11).

7. Ancillary diagnostic tests for tuberculosis – ELISA, etc. recommended for data gathering; see Guidelines for the Control of Tuberculosis in Elephants for current recommendations (2).

8. Other vaccination regimens will depend on regional requirements and exposure risks (consider vaccination for equine encephalitis viruses, Clostridial diseases, Leptospirosis). Insufficient information is available at this time to provide a recommendation for West Nile Virus vaccination of elephants. Contact the SSP veterinary advisor for current information.

**Elephant Serum Bank Submission Form**
**American Zoo and Aquarium Association**
**Elephant Species Survival Plan**

Institution/owner: _____
Submitter: _____
Address: _____
_____
Tel:_____Fax:_____Email: _____

**ANIMAL INFORMATION**
Asian [ ] African [ ] ISIS # _____ Studbook # _____
Name _____ Age:_____ [ ] actual [ ] estimate
Sex: [ ] male [ ] female

**SAMPLE COLLECTION INFORMATION**
Date of sample collection: _____ Time of collection : _____
Site of sample collection: [ ] ear vein [ ]leg vein [ ] other: _____
Health status of animal: [ ] normal [ ] abnormal
Fasted: [ ] no [ ] yes—how long _____
Weight _____ [ ] actual [ ] estimated
Type of restraint: [ ] manual [ ] anesthetized/sedated [ ] behavioral control
Temperament of animal: [ ] calm [ ] active [ ] excited

Type of blood collection tube:
[ ] no anticoagulant (red-top)
[ ] EDTA (purple)
[ ] heparin (green)
[ ] other: _____
Sample handling: [ ]separation of plasma/serum by centrifugation
(check all that apply) [ ] stored as whole blood
[ ] frozen plasma/serum
[ ] other—describe _____

**TB EXPOSURE STATUS**
[ ] Known infected animal
[ ] Known exposure to culture positive source within the past 12 months
[ ] Known exposure to a culture positive source within the past 1–5 years
[ ] No known exposure to a culture positive source in the last 5 years

**TREATMENT INFORMATION**
**Is elephant currently receiving any medication or under treatment? [ ] yes [ ] no**
**If yes, please list drugs and doses:** _____
_____
_____
Time between blood collection and last treatment: _____

---

Ship samples overnight frozen with shipping box marked "PLACE IN FREEZER UPON ARRIVAL"

**Send completed form with samples to:**
Dr. Michele Miller
Disney's Animal Kingdom—Dept. of Vet. Services
P.O. Box 10,000
Lake Buena Vista, FL 32830-1000
(407) 939-7316; email: Michele.Miller@disney.com

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## Appendix

# 15

**Transport Guidelines for Elephants**
**American Zoo and Aquarium Association**
**Elephant Species Survival Plan**
**December 2002**

Elephants can be trained and habituated to transport, with no obvious ill effects. However, zoo elephants are not routinely transported, and planning for movement should be started well in advance. Transport plans require coordination between elephant handlers familiar with the individual elephant to be moved, veterinarians and elephant managers at the sending and receiving institutions, and the contracted transporter, and must comply with local, state, and federal regulations (United States Department of Agriculture). For additional information, refer to the *Elephant Husbandry Manual,* AZA Standards for Elephant Management and Care [Appendix 10, this book], and USDA-APHIS Animal Welfare Act.

The following information should be used as general guidelines when conducting an elephant transport. The final decision for specific procedures should be made in partnership between the shipping and receiving institutions.

### PRIOR TO TRANSPORT

- Transport should be arranged with an individual or company experienced in and properly equipped for moving an elephant. Contact other institutions for references.
- A written transport plan should be developed.
  - Elephant managers and veterinarians from the sending and receiving institutions should be involved.
  - Plan should detail responsibilities of all parties involved.
  - Facilities en route should be contacted in advance for assistance with possible emergencies.
- If an elephant will be transported in a trailer, the trailer should be inspected and meet the following criteria:
  - Allow the elephant to stand comfortably.
  - Provide drainage for urine.
  - Be adequately reinforced and allow the elephant to be safely tethered.
  - Have adequate heating or cooling systems to maintain the temperature between 55 and 70 degrees F with adequate ventilation; if temperatures will be outside this range, the elephant should be monitored more frequently.
  - Permit access for food and water.
  - Allow handlers to adequately monitor the elephant's condition.
- If an elephant will be transported in a crate, it is best to contact other facilities with experience in crate design.
- Elephants shipped by airline must meet the guidelines of the International Air Transport Association (IATA).
- Acclimatization to the trailer or crate may take from 1–6 weeks depending on the individual elephant's temperament. This process should begin as early as possible before the transport date. *Note:* access to the trailer being used for transport may not be feasible if the institution contracts with a private transporter.

### DURING TRANSPORT

- Handlers familiar with the individual elephant should travel with the elephant to the receiving institution.
- A two week supply of hay and grain should accompany the elephant to the new facility. This allows a gradual transition to the new diet.
- The decision to use sedation or chemical immobilization for transport of an elephant should be made in advance as part of the written transport plan.
  - If chemical immobilization or sedation is used to load the animal, the elephant should be held for up to 24 hours or have a veterinarian accompany the shipment to avoid complications associated with drug effects.

- Personnel accompanying the elephant must be familiar with common side effects of the drugs and actions needed to prevent or correct complications. Adequate equipment and supplies should be available.
- If the elephant is being transported by airplane, it is strongly recommended that a veterinarian accompany the elephant. The effects of sedatives or anesthetic drugs combined with the effects of altitude may lead to potentially more serious signs.
- During transport, the elephant should periodically be provided with hay and should be given access to water if the transport time is greater than 16 hours.
- Personnel should regularly monitor the condition of the animal during transport. It is important that adequate ventilation and temperature control be maintained for the comfort and well-being of the elephant.

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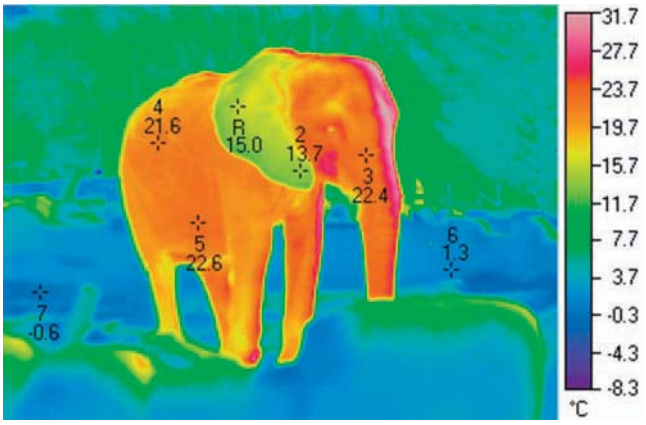



***Figure 13.2.*** *A thermal image of an African elephant taken at an ambient air temperature of 50.7°F (10.4°C). The pinnae are vasoconstricted with only a small difference between the ambient temperature and the temperature of the pinnae (photo by Vaughan Langman, PhD, Shreveport, LA.).*

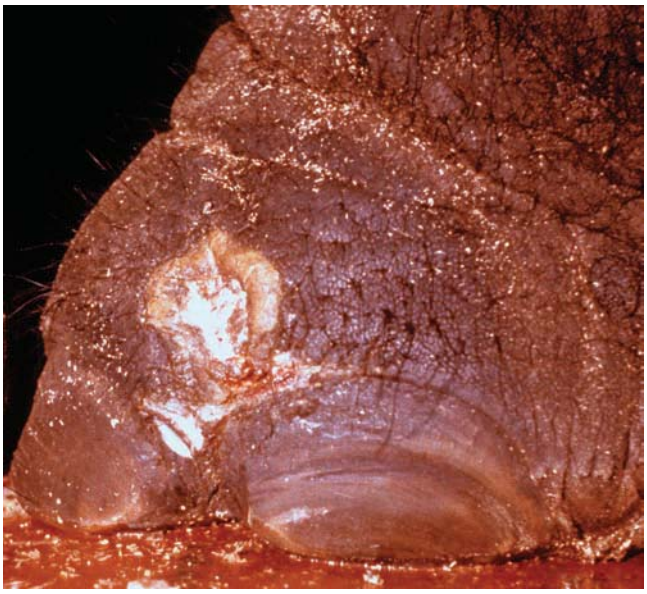


***Figure 14.4.*** *Elephant pox. Large craterlike lesion at the base of two toenails (courtesy of Dr. Kuntze).*

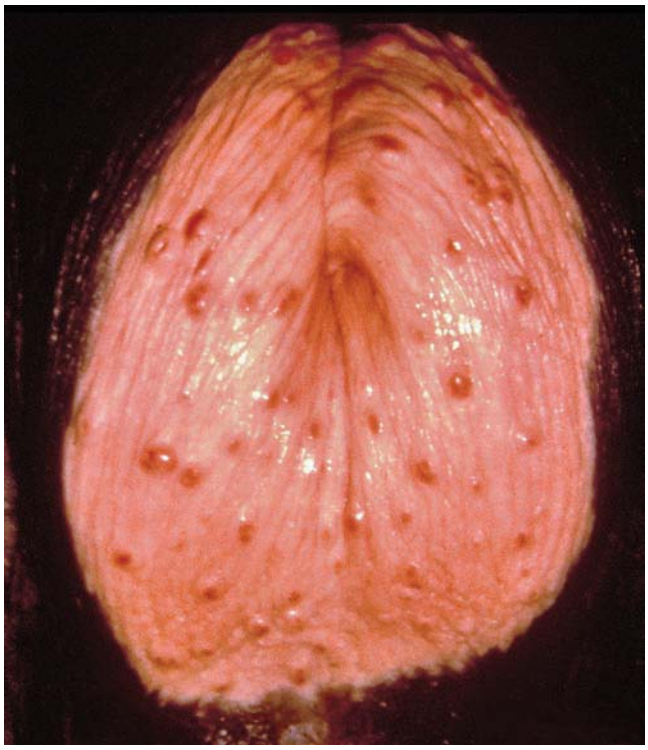


***Figure 14.2.*** *Lymphocytic vulvitis in the distal end of the urogenital canal in an African elephant. These lesions also harbor EEHV (courtesy of Dr. T. Hildebrandt).*

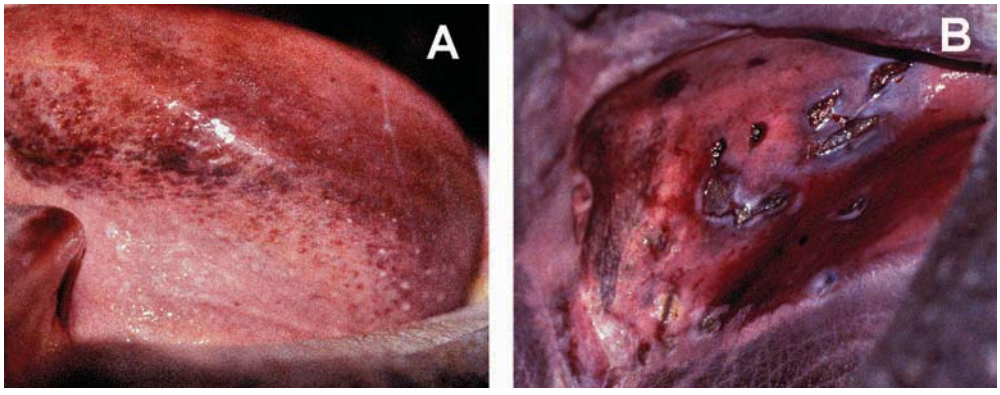



***Figure 14.3.*** *External signs of EEHV. A) adult Asian elephant with tongue cyanosis (dark blue) and mucosal hemorrhages (acknowledgment, Dr. T. Hildebrandt), B) Asian elephant calf with severe palatine ulcers (courtesy of Dr. J. St. Leger).*

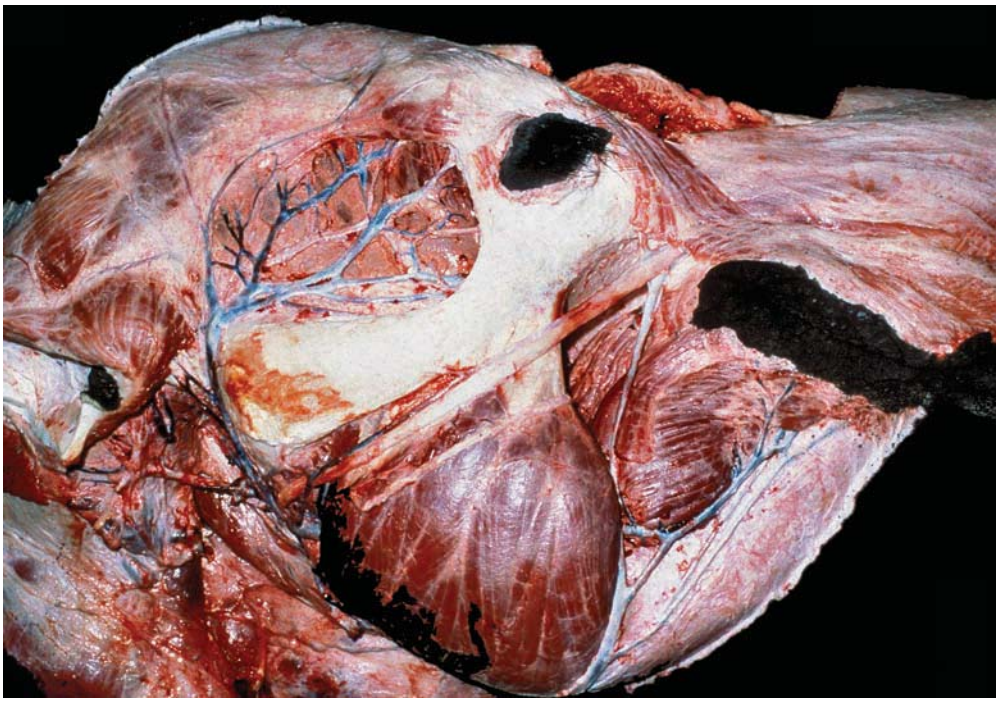


***Figure 14.5.*** *Temporal or musth gland from an Asian elephant calf. Dissected surface shows prominent venous circulation, which outlines the gland (courtesy of Dr. Don Frey).*

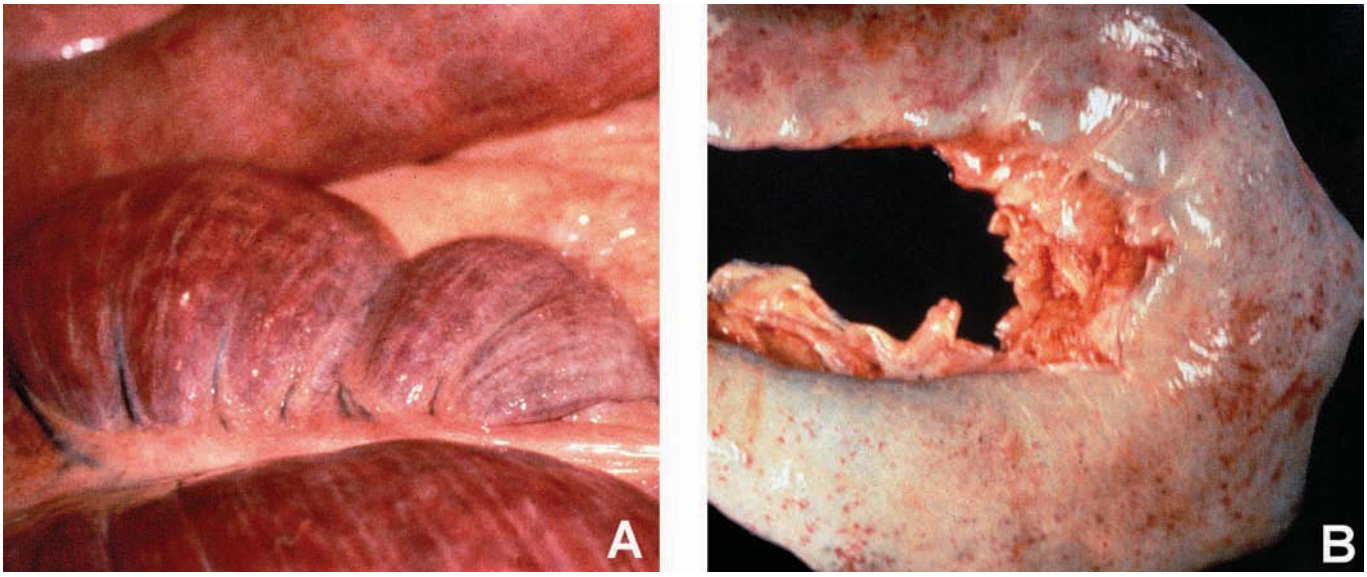



***Figure 14.7.*** *Intrabdominal hemorrhages in Asian elephant calf with EEHV. A) diffuse involvement of large intestinal serosae, B) multifocal involvement of splenic capsule.*

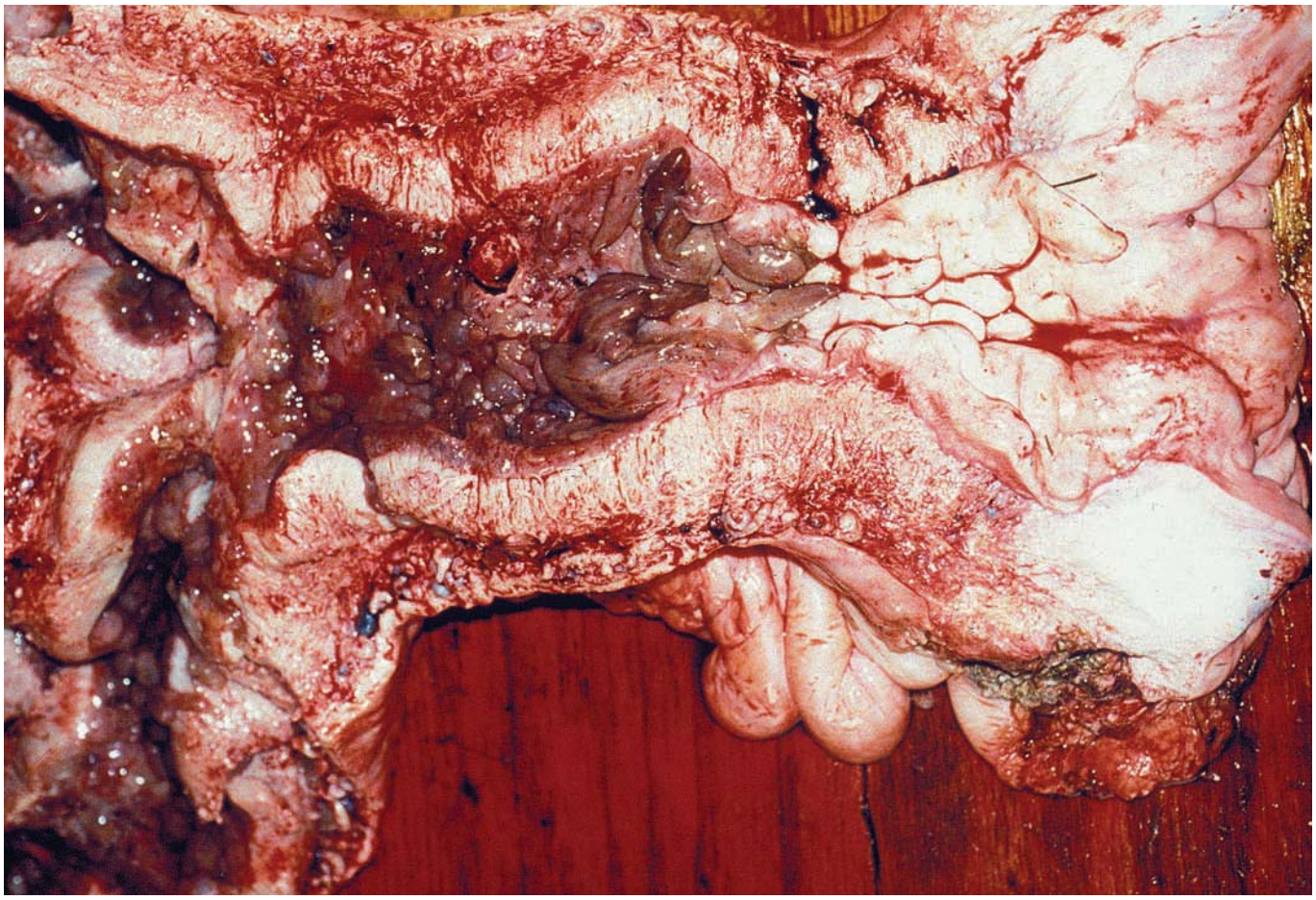


***Figure 14.8.*** *Uterus (body with cervix to the right) of an elderly Asian elephant with cystic endometrial hyperplasia.*

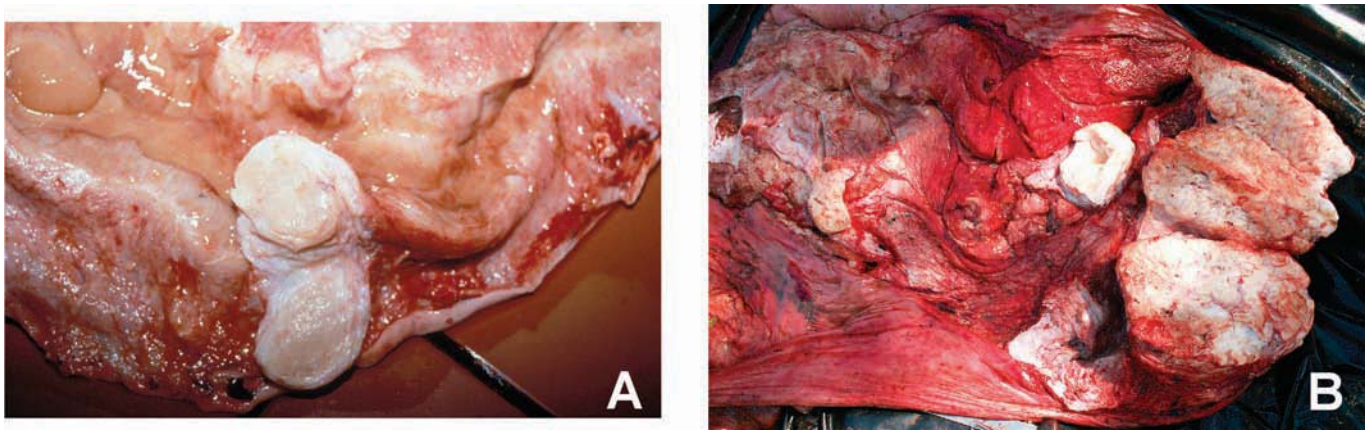



***Figure 14.9.*** *Elderly Asian elephants with uterine leiomyomas. A) mild involvement with scattered tumor masses within the myometrium (courtesy of Dr. T. Hildebrandt), B) massive uterine involvement in another elephant with large confluent fibrous-appearing tumor masses.*

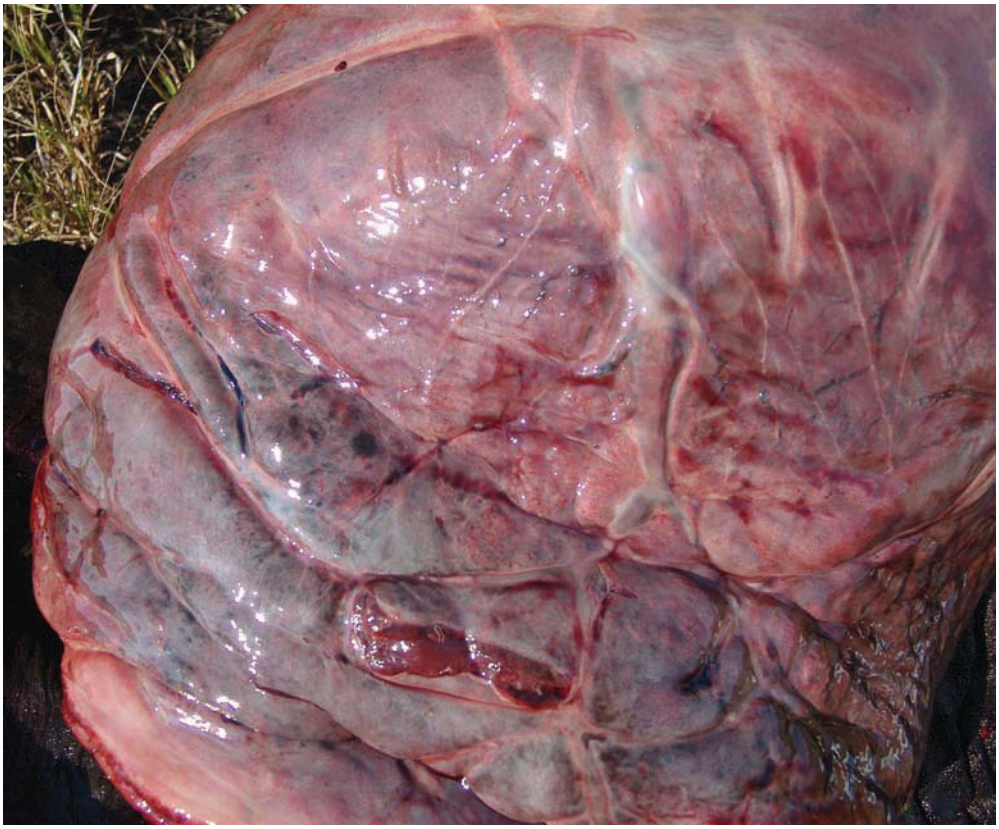


***Figure 14.10.*** *Epicardial hemorrhages in the heart of an African elephant that died of encepahlomyocarditis virus (courtesy of D. F. Keet).*

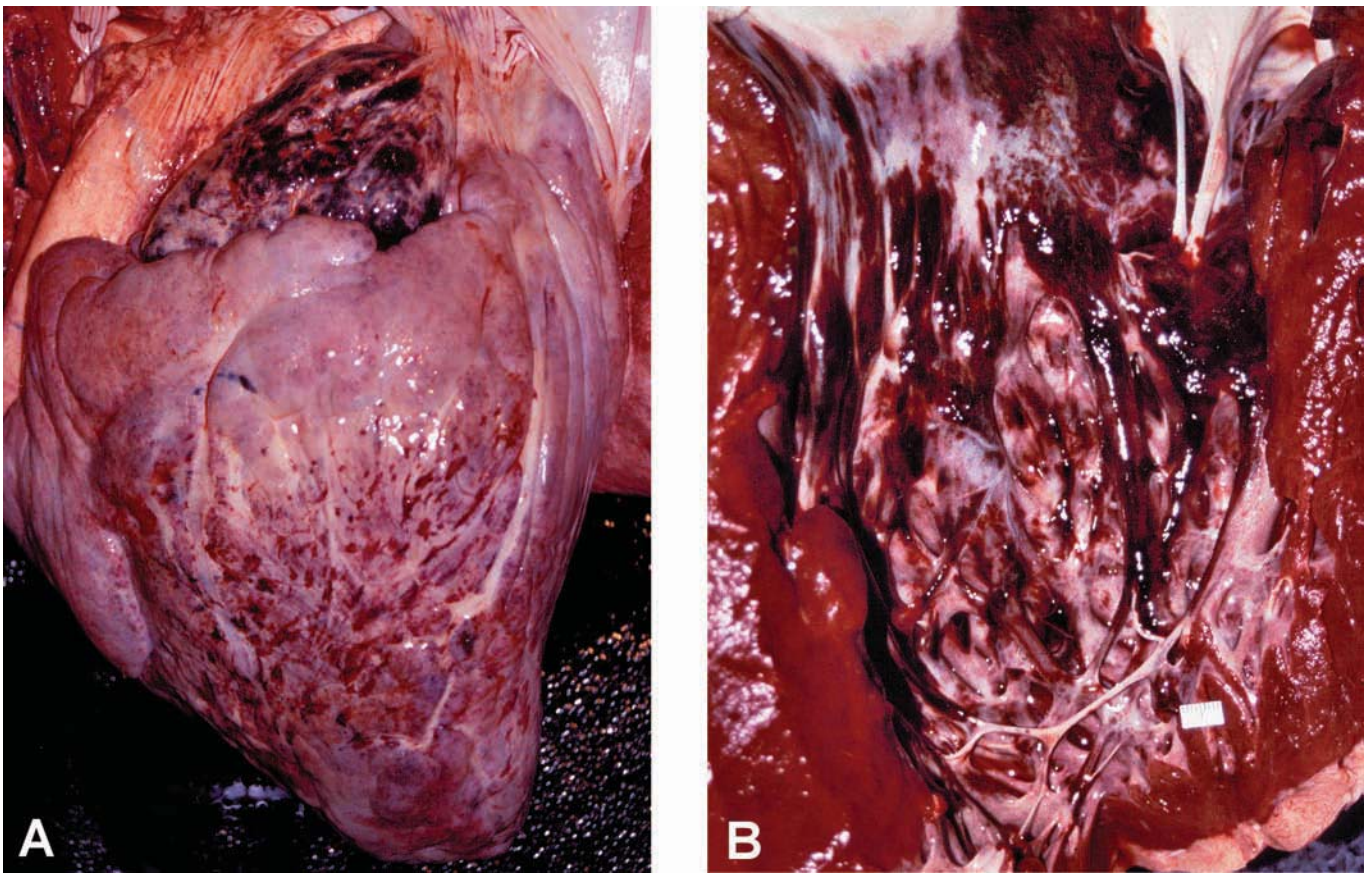



***Figure 14.11.*** *Heart from Asian elephant calf with EEHV (acknowledgment, Dr. J. St. Leger). A) prominent epicardial hemorrhages base of heart and ventricular surfaces, B) extensive endocardial hemorrhages.*

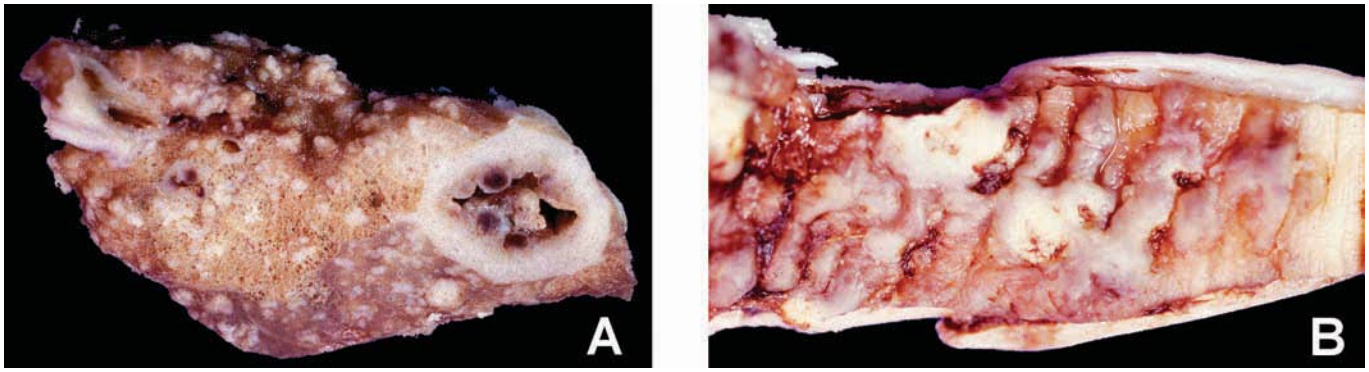



***Figure 14.12.*** Mycobacterium tuberculosis *infection in an Asian elephant. A) lung segment with small bronchial and parenchymal tuberculous granulomas, B) tracheal segment with tuberculous mucosal plaques.*

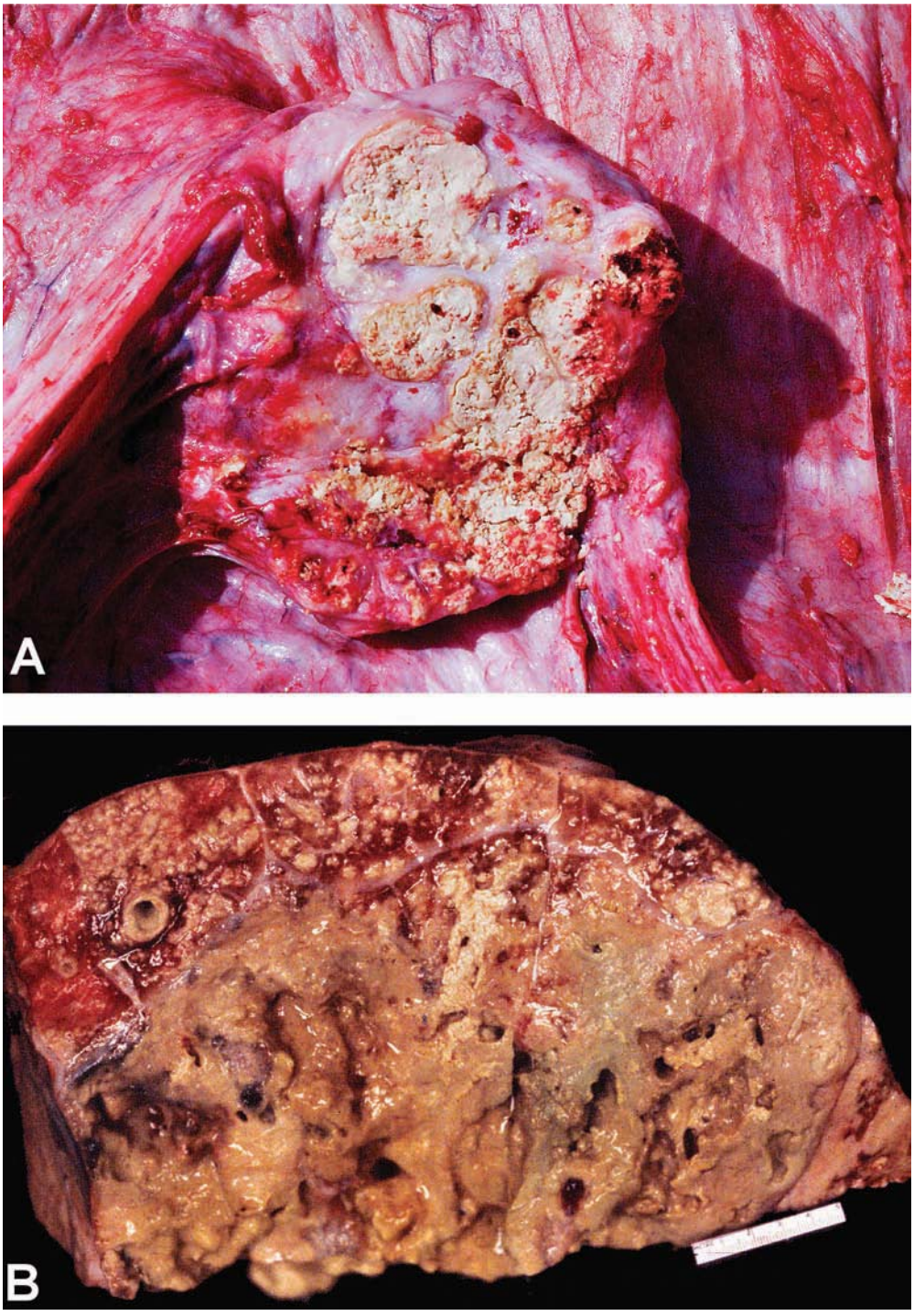



***Figure 14.13.*** *Advancing* Mycobacterium tuberculosis *infection in Asian elephants. A) segment of lung from elephant with caseonecrotic lesions. B) massive caseocalcareous tuberculous pneumonia in another elephant (courtesy of Rev Sci Tech Off Int Epiz 20(1):291–303, 2001, and Dr. H. Kinde).*

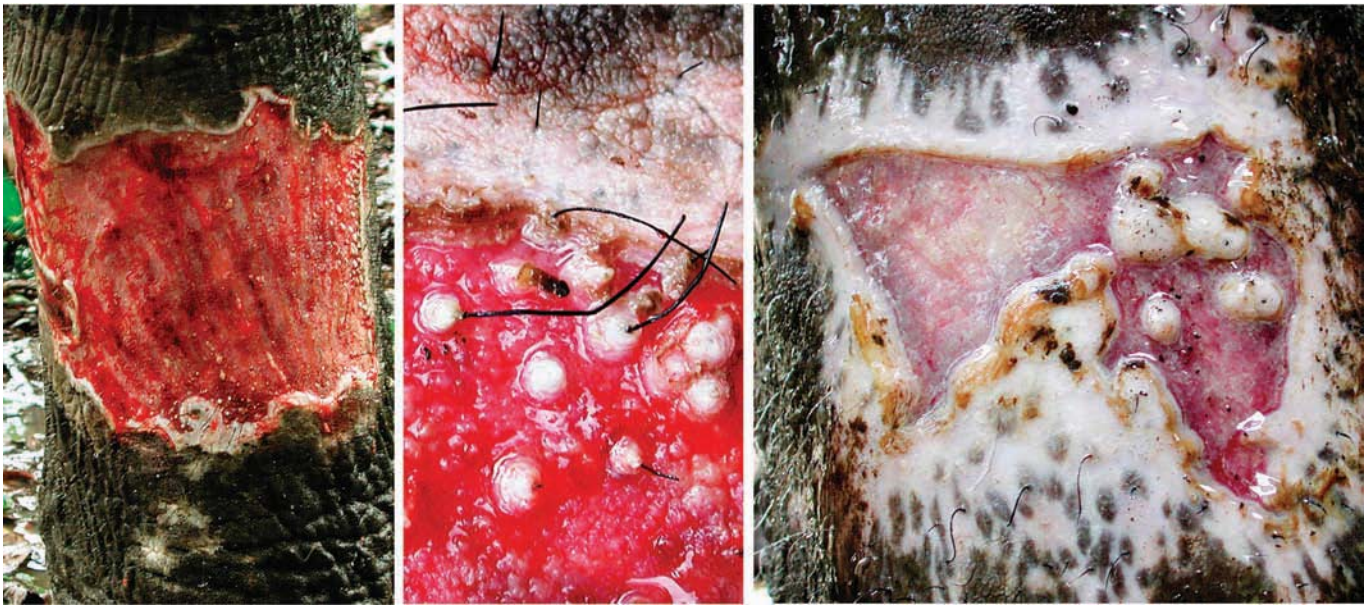


***Figure 18.3.*** *Healing leg wound. The center photo is a close-up of the granulation bed. The multifocal beds of granulation tissue will coalesce to close the wound (photo by Hank Hammatt).*

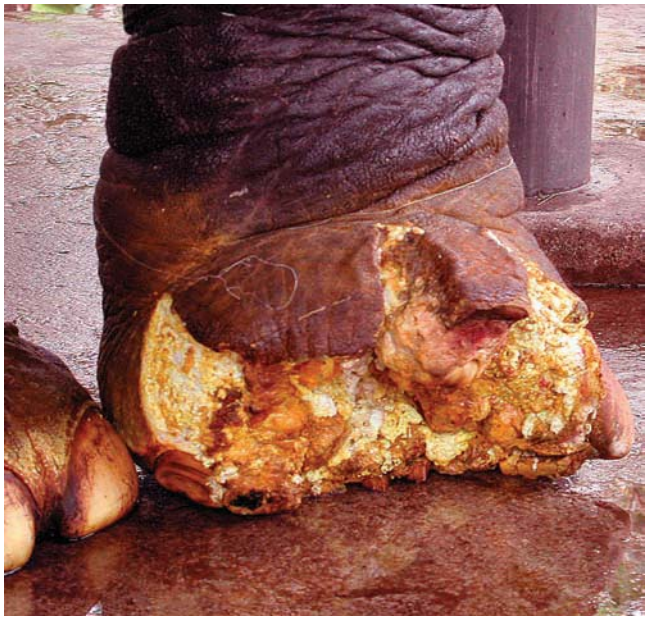
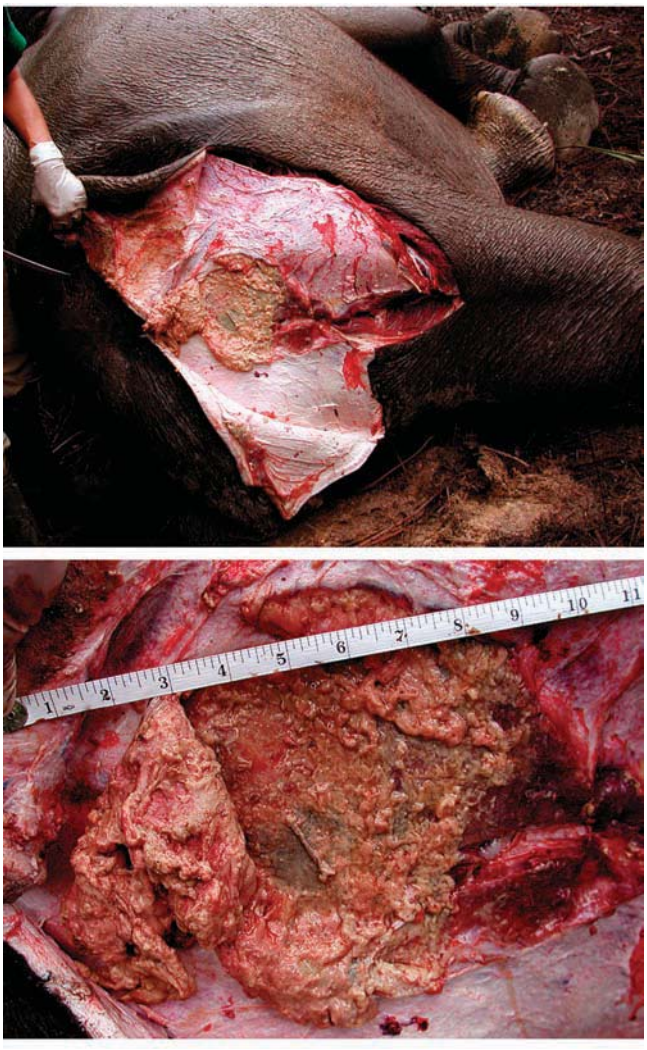


***Figure 18.5.*** *Severe injury to skin and soft tissue resulting from a land mine injury (photo by Hank Hammatt).*



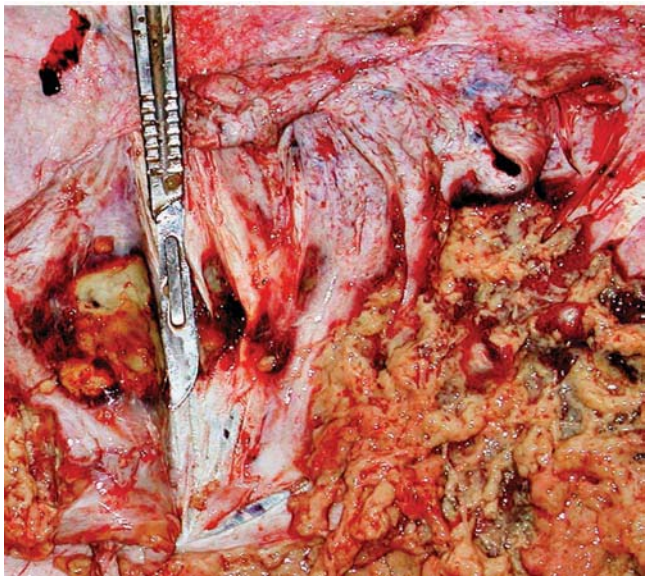


***Figure 18.7.*** *Necrotizing fasciitis subsequent to an untreated dart wound (photo by Hank Hammatt).*

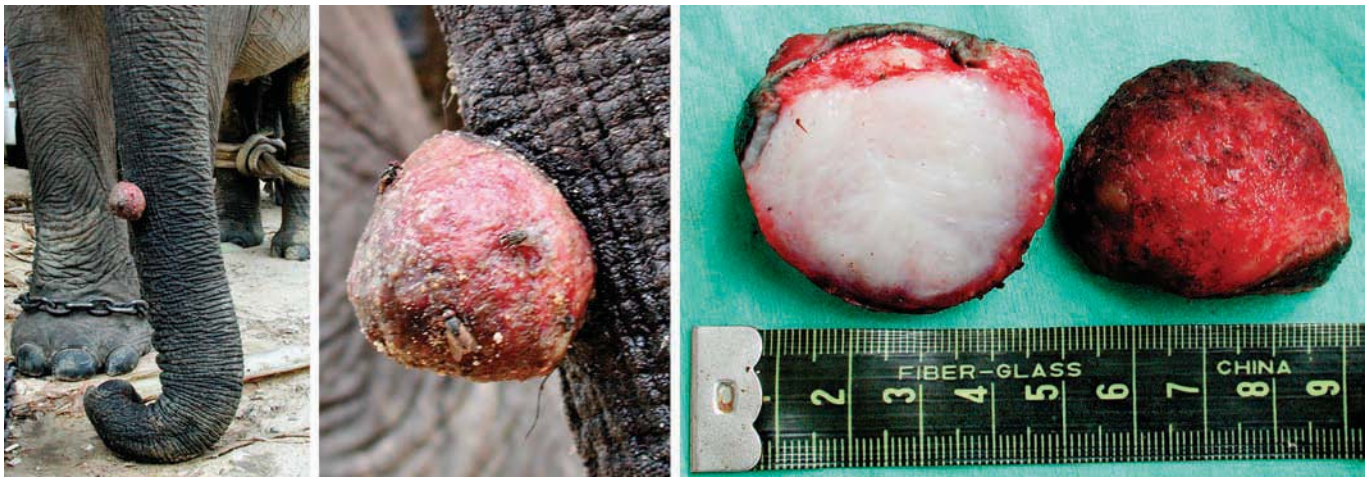



***Figure 18.8.*** *Fibrosarcoma in a 4-year-old Asian elephant (photo by Hank Hammatt).*

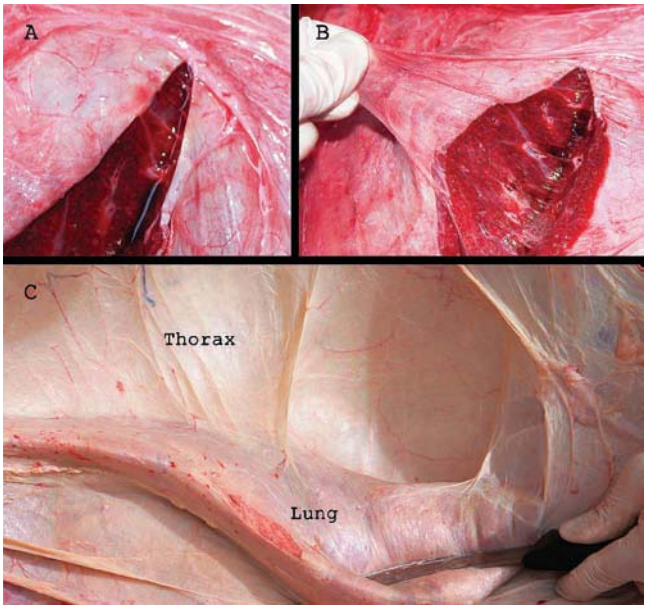



***Figure 21.3.*** *Three photographs of normal pleural space connective tissue (PSCT) from an Asian elephant: A) close-up view of the relaxed, gelatinous PSCT resting on the lung surface; B) a portion of the PSCT is pulled away from the lung surface; C) lung is pulled and cut away from the thoracic wall, showing the extremely stretched PSCT as it is typically seen during necropsy.*

*Note:* All slides were stained with Wright's stain. These images are presented to aid in the identification of the cells commonly encountered in elephant blood. The original photomicrographs may have been manipulated (cropped) to feature individual cells. The magnification at which the original photomicrograph was taken is therefore not included. The figures that include multiple cells may also have been enlarged; however, the relative sizes of the cells is not altered and is consistent with what would be viewed through the microscope.

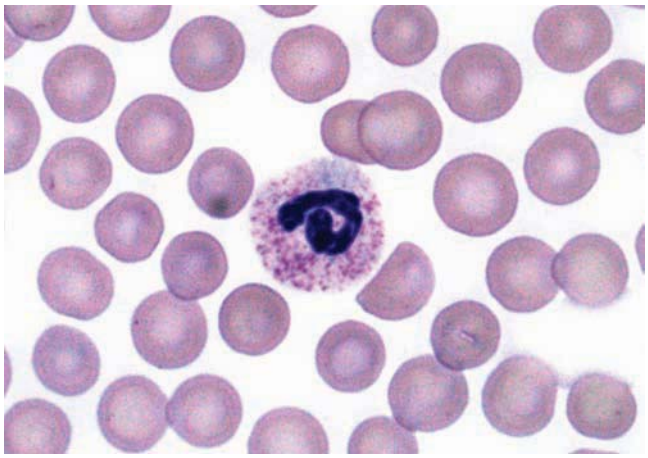


***Figure 25.3.*** *Heterophil.*

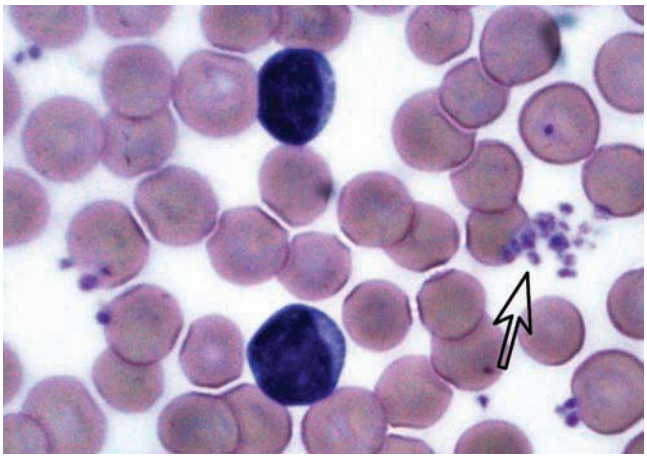


***Figure 25.4.*** *Two lymphocytes. Note clump of platelets (arrow) and other platelets scattered in field.*

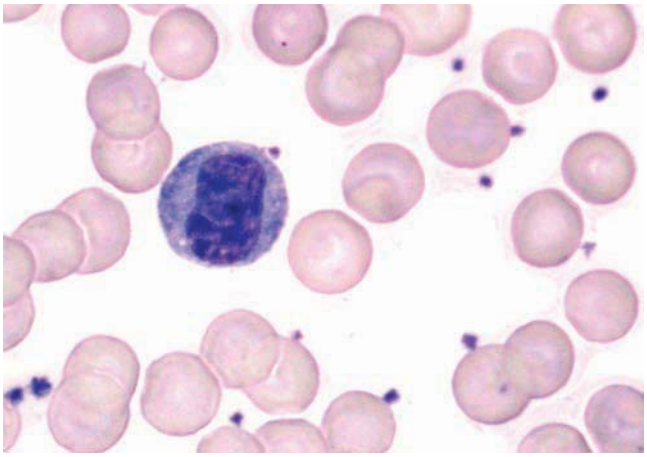


***Figure 25.5.*** *Unsegmented (classic) monocyte.*

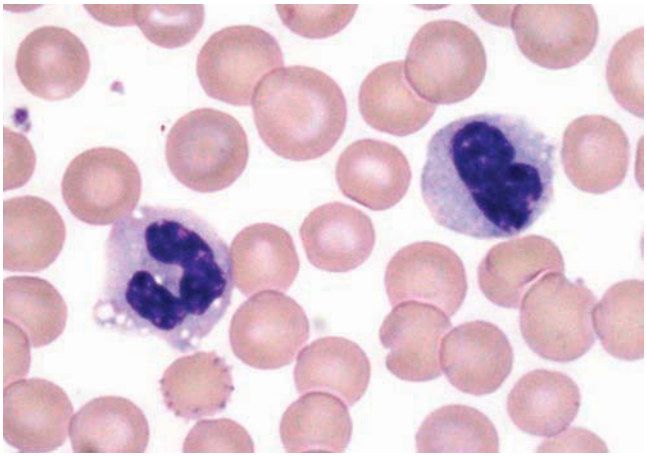


***Figure 25.6.*** *Segmented monocytes (bilobed and trilobed).*

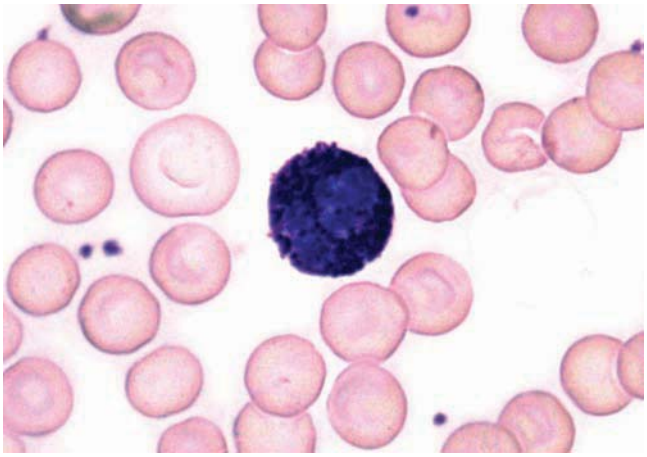


***Figure 25.7.*** *Basophil.*

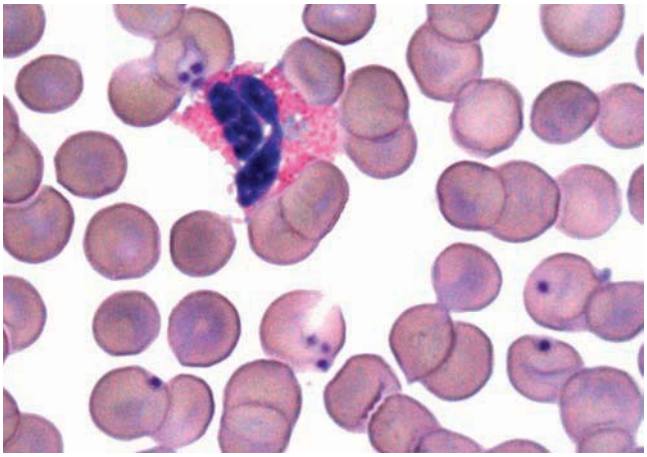


***Figure 25.8.*** *Eosinophil.*

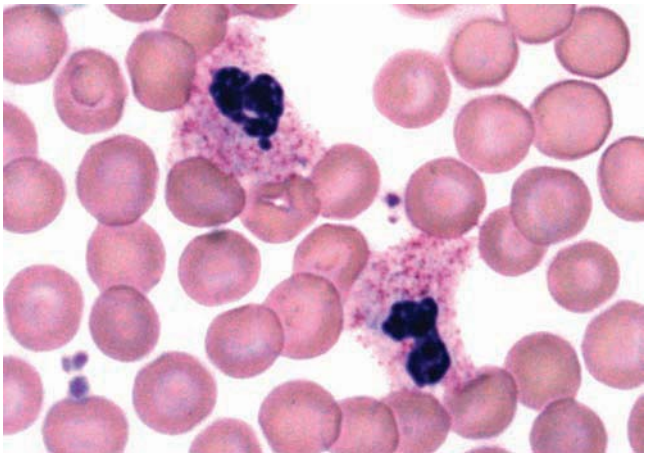


***Figure 25.9.*** *Two heterophils.*

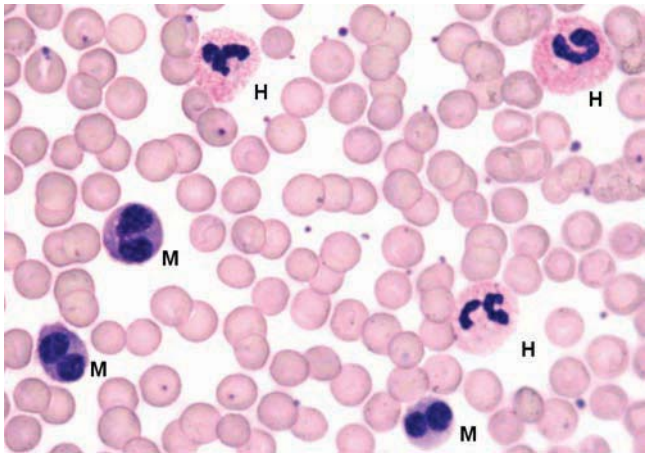



***Figure 25.10.*** *Three heterophils (H) and three bilobed monocytes (M).*

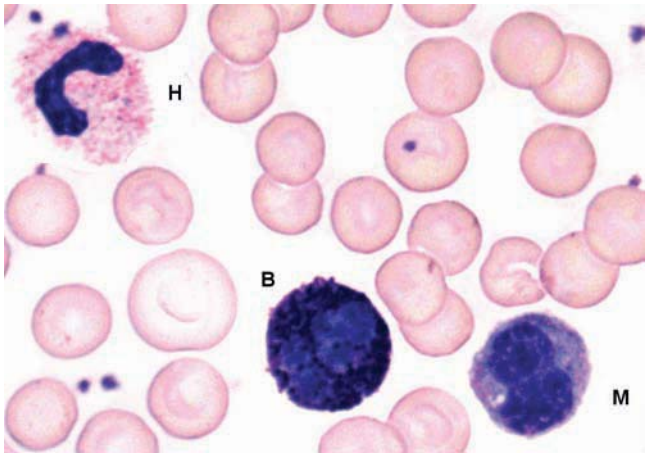



***Figure 25.11.*** *Heterophil (H), basophil (B), and bilobed monocyte (M).*

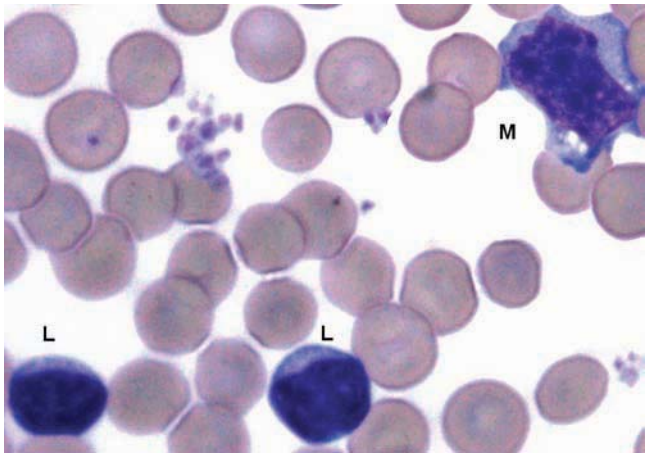



***Figure 25.12.*** *Unsegmented (classic) monocyte (M) and two lymphocytes (L).*

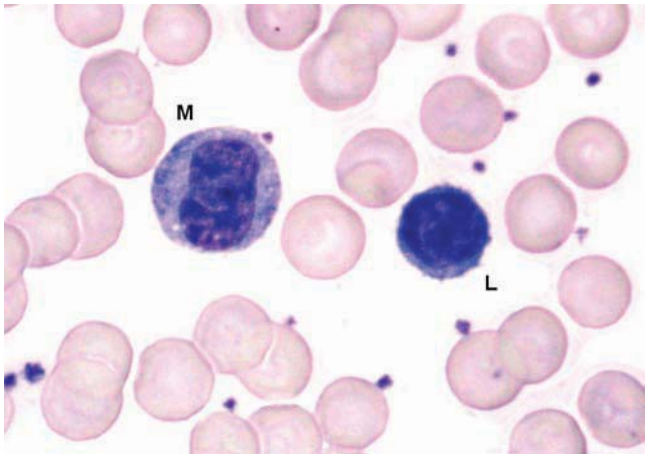



***Figure 25.13.*** *Unsegmented (classic) monocyte (M), lymphocyte (L), and scattered platelets.*

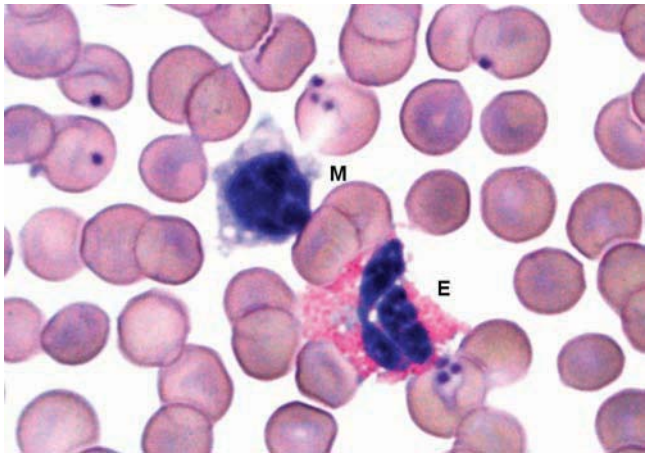



***Figure 25.14.*** *Eosinophil (E). The cell labeled M presents an identification dilemma. It could be identified as an unsegmented (classic) monocyte based on the following features: 1) it has more cytoplasm than the other lymphocytes and 2) it has two small cytoplasmic granules at about 7 o'clock. Alternatively it could be identified as a lymphocyte based on these features: 1) it has a heavily condensed nucleus and 2)it has less cytoplasm than the other monocytes. Cells of this type are rare and unlikely to result in significant error on the differential count.*

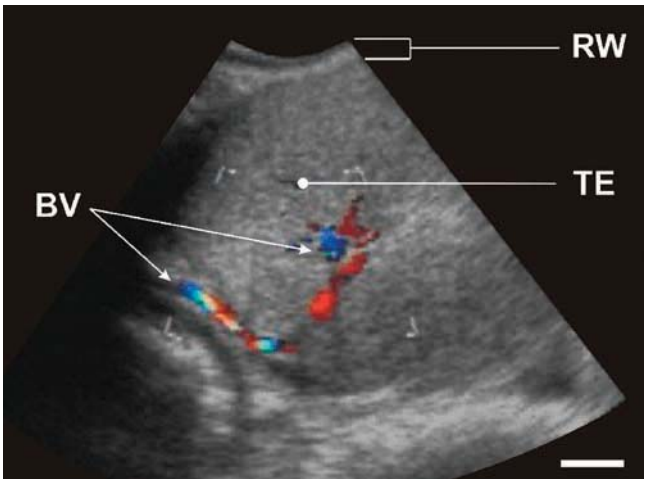
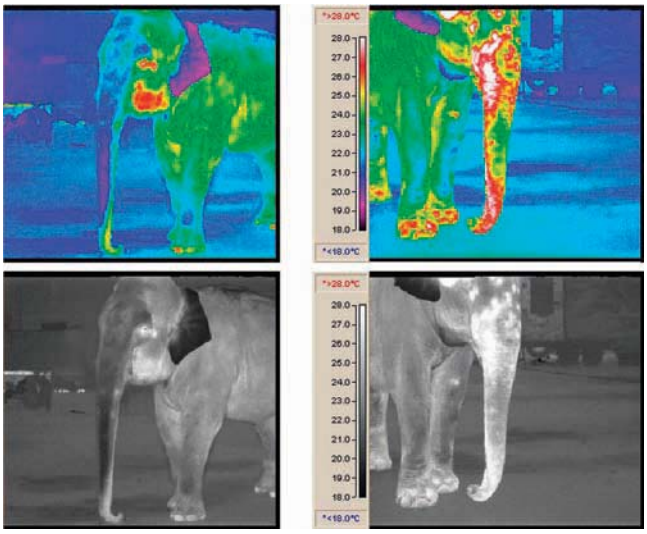



***Figure 27.24.*** *Transrectal color-flow Doppler image (5-2 MHz) of an active right testis (TE) in a breeding bull. Note the intratesticular blood vessels (BV) visualized by this angio-mode. The rectal wall (RW) appears as a moderate echoic strip on top of the sonogram.*



**A** **B**

***Figure 30.1.*** *(A) Thermal signature of a 45-year old Asian elephant with a 5-year history of decreased trunk use. The trunk is cooler on both the cranial and caudal surfaces when compared to the unaffected herdmate (B) (thermal image courtesy of Dr. Gregory Fleming).*

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## Murray E. Fowler and Susan K. Mikota

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**Murray E. Fowler** is the author of the bestselling *Zoo and Wild Animal Medicine, Fifth Edition*. He has also written *Medicine and Surgery of South American Camelids*, *Restraint and Handling of Wild and Domestic Animals*, and *Biology and Surgery of South American Wild Animals*. He is currently Professor Emeritus of Zoological Medicine, University of California-Davis. For the past four years he has been a part-time employee of Ringling Brothers, Barnum and Bailey's Circus.

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